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BACKGROUND: The traditional predictors of the outcome of weaning from mechanical ventilation—minute ventilation (Vt) and maximal inspiratory pressure (Pimax)—are frequently inaccurate. We developed two new indexes: The first quantitates rapid shallow breathing as the ratio of respiratory frequency to tidal volume (f/Vr), and the second is termed CROP, because it integrates thoracic compliance, respiratory rate, arterial oxygenation, and Pimax.

METHODS: The threshold values for each index that discriminated best between a successful and an unsuccessful outcome of weaning were determined in 36 patients, and the predictive accuracy of these values was then tested prospectively in an additional 64 patients. Sensitivity and specificity were calculated, and the data were also analyzed with receiver-operating-characteristic (ROC) curves, in which the proportions of true-positive results and false-positive results are plotted against each other for a number of threshold values of an index: the area under the curve reflects the accuracy of the test.

RESULTS: Sensitivity was highest for Pimax (1.00), followed closely by the f/Vr ratio (0.97). Specificity was highest for the f/Vr ratio (0.64) and lowest for Pimax (0.11). The f/Vr ratio was the best predictor of successful weaning, and Pimax and the f/Vr ratio were the best predictors of failure. The area under the ROC curve for the f/Vr ratio (0.89) was larger than that under the curves for the CROP index (0.78, p < 0.05), Pimax (0.61, p < 0.001), and Vt (0.40, p < 0.001). CONCLUSIONS: Rapid shallow breathing, as reflected by the f/Vr ratio, was the most accurate predictor of failure, and its absence the most accurate predictor of success, in weaning patients from mechanical ventilation.


We have documented the physiologic effects of fiberoptic bronchoscopy with bronchoalveolar lavage (BAL) and endobronchial biopsy performed under local anesthesia in 20 asthmatic subjects, 8 healthy nonatopic control subjects, and 8 atopic nonasthmatic subjects. Premedication consisted of nebulized albuterol (2.5 mg; except for the study of atopic nonasthmatic subjects), ipratropium bromide (500 μg), and intramuscular atropine (0.6 mg). Intravenous midazolam was given for mild sedation, and oxygen was delivered via a nasal cannula. FEV1 was measured before and after premedication, immediately postbronchoscopy, and after 2 h recovery. There was a significant fall
in mean (± SD) FEV₁, immediately postbronchoscopy in both the asthmatic (26.2 ± 16.7%; p < 0.001) and normal (9 ± 4.7%; p < 0.05) groups, which in the asthmatic subjects correlated inversely with the concentration of methacholine provoking a 20% fall in FEV₁ (PC₂₀) measured 5 days prebronchoscopy (r = -0.74, p < 0.001) but not with symptom scores, albuterol use, or peak expiratory flow (PEF) variation recorded during 2 wk before the investigation. There was significant arterial hemoglobin O₂ desaturation during biopsy in the asthmatic subjects (median 3%, range 1-17% fall from baseline; p < 0.01), which was not related to any of the measured indices of asthma severity. PC₂₀, measured 5 days before and 5 days after bronchoscopy in the asthmatic subjects and 2 days before and 1 day after bronchoscopy in the atopic nonasthmatic subjects was not significantly affected by the procedure. We conclude that fiberoptic bronchoscopy with BAL and endobronchial biopsy can be conducted safely in asthmatic subjects, but requires caution in those with very responsive Airways. Prior measurement of PC₂₀ and oximetry monitoring throughout the procedure are strongly recommended.


Data collected on 12- to 74-year-old whites (n = 10,854) during the second National Health and Nutrition Examination Survey, 1976 to 1980, a sample of the U.S. population, were used to determine the association between various respiratory symptoms and the degree of allergen skin test reactivity. Prick-puncture testing using...
eighth unstandardized allergens was performed. Allergen skin test reactivity was classified by means of the mean diameter of the erythema reaction at the 20-minute reading. Nonreactors were the comparison group. The prevalence of allergic rhinitis increased as allergen skin test reactivity increased, with the odds ratio exceeding 8 for the group with two or more positive test results. The prevalence of asthma increased with increasing allergen skin test reactivity only in nonsmokers. The odds ratio for allergic rhinitis with allergen skin test reactivity was higher with outdoor than indoor allergens. The association of allergic rhinitis with allergen skin test reactivity was higher when a physician had previously diagnosed allergic rhinitis. Chronic rhinitis was not associated with allergen skin test reactivity.


Six pulse oximeters with finger probes were studied in three groups of 17 hypoxemic patients with COPD aged 50 to 75 years. Transcutaneous arterial oxygen saturation (SpO2) was measured with the Nellcor N101 (oximeter 1a), the Ohmeda Biox III (oximeter 1b), the Nellcor N200 (oximeter 2a), the Critikon Oxyshuttle (oximeter 2b), the Radiometer Oxi100 (oximeter 3a), and the Ohmeda Biodex 3700 (oximeter 3b). The S5O2 was compared with S5O2 measured in simultaneously withdrawn samples of arterial blood (Radiometer OSM2) at three 20-minute steady state levels of Fio2 ranging from 0.21 to 0.40 (S5O2, 62 to 100%). The bias (mean S5O2-S5O2 difference) and the error in precision (SD of the differences) were both below 4% for instruments 1a and 1b and remained below 1.2% and 3%, respectively, for the others. A good agreement between S5O2 and S5O2, as reflected by the Bartko intraclass correlation coefficient, was observed in instruments 2a, 3a, and 3b. The individual relationships between S5O2-S5O2 differences and S5O2 appeared to be linear and parallel. With four instruments (1a, 1b, 2a, and 2b), the mean slope of this relationship was negative, showing a systematic instrumental error: the lower the S5O2, the larger the overestimation of S5O2. The scattering of the data (precision) principally reflects a subject source of error. In most instruments a technical adjustment could greatly improve instrumental errors and accuracy. The correction of the errors due to between-subject variation would require a system of calibration adjustable by the users to each individual.


Circuits on mechanical ventilators with cascade humidifiers are routinely changed every day or every other day, although humidifying cascades have been considered unlikely to increase the risk of respiratory infection because they do not generate aerosols. Moreover, changing ventilator tubing every 24 rather than every 48 h increases the risk of ventilator-associated pneumonia. To study the effects of ventilator circuit changes on the rate of nosocomial pneumonia and on patient and circuit colonization, 73 consecutive patients requiring continuous mechanical ventilation for more than 48 h were randomly assigned to either ventilator circuit changes every 48 h (Group 1, n = 38) or no change (Group 2, n = 35). Patients dying or being weaned before 96 h were not analyzed (Group 1, n = 3; Group 2, n = 7; leaving Group 1, n = 35, and Group 2, n = 28: p = 0.13). Ventilator-associated pneumonia was defined as the occurrence during mechanical ventilation or within 48 h after weaning of a new and persistent infiltrate on chest x-ray, purulent tracheal secretions, and a positive culture of a protected brush specimen (≥ 106 cfu/mL). Bacterial colonization was assessed every 48 h by quantitative cultures of pharyngeal swab, tracheal aspirate, humidifying cascade, and expiratory tubing trap. The two groups were similar in terms of age, indication for and duration of ventilation, and severity of illness. The incidence of pneumonia was similar in both groups (11 of 35 and 8 of 28 in Groups 1 and 2, respectively: p = 0.8), as was the duration of ventilation before pneumonia (10.1 ± 5.8 versus 9.1 ± 2.9 days: p = 0.7). The level of colonization by both gram-positive and gram-negative bacteria was the same in both groups. We conclude that not changing ventilator circuits during mechanical ventilation has no adverse effect on the rate of nosocomial pneumonia or on patient and circuit colonization. Substantial savings in expenses of tubing and personnel time could be obtained without apparent adverse effect.


Many infants admitted to neonatal intensive care units are the children of women infected with the human immunodeficiency virus (HIV); they have approximately a 30% risk of infection. To investigate attitudes surrounding treatment for such newborns, we conducted a survey of
professionals at six neonatal intensive care units in New York City. A significant proportion of the 247 respondents recommended less aggressive treatment for non-HIV-related conditions for infants at risk for HIV compared with those not at risk. For example, 97% of respondents recommended open heart surgery for an infant with no known HIV risk, but only 77% recommended surgery for an infant whose mother had acquired immunodeficiency syndrome; if certain the infant was infected, 42% of respondents recommended surgery. We conclude that perceived HIV status may influence decision making about treatment for non-HIV-related conditions for critically ill patients, including infants not actually infected. Ethical issues concerning the relevance of HIV status need to be examined.


We evaluated the conditions of 33 patients who completed an outpatient pulmonary rehabilitation program to determine what types of improvements occurred, and whether these changes were related to the baseline degree of ventilatory impairment, to determine whether rehabilitation was beneficial to patients, regardless of the degree of underlying lung dysfunction. Endurance measurements, including sustained submaximal performance on a cycle ergometer and the 12-min walk distance (1.349 ± 625 feet to 1.700 ± 670 feet) increased significantly (p < 0.01), as did multiple educational and subjective parameters. Maximal exercise performance on a graded cycle test improved very little, with a decline in the ventilatory equivalent for oxygen consumption (Ve/VO2) being the only significant change (48.2 ± 28.3 L/min to 36.6 ± 8.7 L/min). Of the observed changes, only one endurance measurement, the sustained submaximal exercise performance, correlated with FEV1, (r = 0.5, p < 0.01), but only if it was expressed as an absolute number (liters) and not as percent predicted. Lung function did not correlate with changes in the 12-min walk distance, in maximal exercise performance on the cycle ergometer, or with changes in educational and subjective parameters. We conclude that because the magnitude of change in both physiologic and physiologic parameters was not directly related to lung function, the benefits of rehabilitation can extend to all patients with chronic lung disease, regardless of the severity of pre-existing pulmonary dysfunction.


STUDY OBJECTIVES: Survival from out-of-hospital cardiac arrest in cities with populations of more than 1 million has not been studied adequately. This study was undertaken to determine the overall survival rate for Chicago and the effect of previously reported variables on survival, and to compare the observed survival rates with those previously reported. DESIGN: Consecutive prehospital arrest patients were studied prospectively during 1987. SETTING: The study area was the city of Chicago, which has more than 3 million inhabitants in 228 square miles. The emergency medical services system, with 55 around-the-clock ambulances and 550 paramedics, is single-tiered and responds to more than 200,000 emergencies per year. TYPE OF PARTICIPANTS: We studied 3,221 victims of out-of-hospital cardiac arrest on whom paramedics attempted resuscitation. MEASURE-MENTS AND MAIN RESULTS: 91% of patients were pronounced dead in emergency departments, 7% died in hospitals, and 2% survived to hospital discharge. Survival was significantly greater with bystander-witnessed arrest, bystander-initiated CPR, paramedic-witnessed arrest, initial rhythm of ventricular fibrillation, and shorter treatment intervals. CONCLUSIONS: The overall survival rates were significantly lower than those reported in most previous studies, all based on smaller communities; they were consistent with the rates reported in the one comparable study of a large city. The single factor that most likely contributed to the poor overall survival was the relatively long interval between collapse and defibrillation. Logistical, demographic, and other special characteristics of large cities may have affected the rates. To improve treatment of cardiac arrest in large cities and maximize the use of community resources, we recommend further study of comparable metropolitan areas using standardized terms and methodology. Detailed analysis of each component of the emergency medical services system will aid in making improvements to maximize survival of out-of-hospital cardiac arrest.


Although aerosolized pentamidine (AP) has recently been approved for prophylaxis and is undergoing clinical trials for treatment of pneumocystis pneumonia (PCP), factors important in the deposition of AP have not been described. Using radioaerosol techniques, deposition was measured in 22 patients receiving AP for prophylaxis or treat-
ment of PCP. In all patients total and regional deposition of pentamidine, breathing pattern, pulmonary function (PFT), regional ventilation, and type of nebulizer were analyzed. Bronchoalveolar lavage (BAL) was performed 24 h after inhalation to assess the relationship between pentamidine levels in BAL fluid and measured aerosol deposition. The nebulizers tested were the Marquest Respirgard II and the Cadema Aerotech II, both previously characterized in our laboratory. The aerosol particles consist of water droplets containing dissolved pentamidine and technetium 99m bound to albumin. Analysis of particles sampled during inhalation via cascade impaction confirmed a close relationship between radioactivity in the droplets and the concentration of pentamidine as measured by HPLC (r = 0.971, p < 0.0001; n = 18). Deposition was measured by capturing inhaled and exhaled particles on absolute filters and measuring radioactivity. This technique allows the determination of the deposition fraction (DF, the fraction of the amount inhaled that is deposited), which provides information on factors strictly related to the patient. To confirm the filter measurements, pentamidine deposition was also measured by gamma camera. The camera measurement was possible because each patient’s thoracic attenuation of radioactivity was determined by a quantitative perfusion scan (mg pentamidine deposited via both techniques, r = 0.949, p < 0.0001; n = 26). Regional lung volume and ventilation were determined by xenon 133 equilibrium scan and washout. Pentamidine deposition varied markedly between patients, but BAL levels of pentamidine significantly correlated with measured deposition (r = 0.819, p < 0.01; n = 9). DF averaged 0.621 ± 0.027 (SEM) and did not correlate with any measured lung parameter, including breathing pattern and PFT.

Regional deposition did not correlate with regional ventilation. The major factor influencing pentamidine deposition was aerosol delivery (mg deposited versus mg inhaled; r = 0.963, p < 0.0001; n = 26). The nebulizer was an important determinant of aerosol delivery, with the Aerotech delivering between 2.5 and 5 times more drug than the Respirgard. These observations are important in assessing treatment failure and cost of therapy.
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**Dramatic reductions in neonatal morbidity and mortality reported in clinical trials**
Improvement in clinical outcome after EXOSURF Neonatal has been significant in infants at risk of developing RDS as well as those with established RDS. Prophylactic as well as rescue treatment with EXOSURF Neonatal has dramatically reduced morbidity and mortality in infants weighing greater than 700 grams.

### SIGNIFICANT REDUCTIONS IN OVERALL MORTALITY FROM ANY CAUSE IN MIDDLE-SIZE AND LARGE INFANTS

(Percent reductions with EXOSURF)

<table>
<thead>
<tr>
<th>Age</th>
<th>Prophylactic treatment</th>
<th>Rescue treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>700-1100 grams (N=446)</td>
<td>700-1100 grams (N=716)</td>
</tr>
<tr>
<td>&lt;10 Days</td>
<td>—</td>
<td>59%</td>
</tr>
<tr>
<td>&lt;28 Days</td>
<td>40%*</td>
<td>44%</td>
</tr>
<tr>
<td>≤1 Year</td>
<td>44%</td>
<td>41%</td>
</tr>
</tbody>
</table>

\*P<0.01; \*P<0.001. N=Number of infants enrolled in the clinical trials.

A single prophylactic dose of EXOSURF Neonatal reduced 1-year mortality by 44%. Two additional prophylactic doses provided an additional 41% reduction in 1-year mortality.

### SIGNIFICANT REDUCTIONS IN MORTALITY FROM RDS IN MIDDLE-SIZE AND LARGE INFANTS

(Percent reductions with EXOSURF)

<table>
<thead>
<tr>
<th>Reduction in death from RDS</th>
<th>Prophylactic treatment</th>
<th>Rescue treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>700-1100 grams (N=446)</td>
<td>700-1350 grams (N=419)</td>
</tr>
<tr>
<td>58%*</td>
<td>66%*</td>
<td>63%</td>
</tr>
</tbody>
</table>

\*P<0.01. N=Number of infants enrolled in the clinical trials.
Rapid onset of action documented in rescue use

Improvements in mean FiO₂ and mean alveolar-arterial PO₂ (A-a) gradient were present by 2 hours after dosing in middle-size babies (700-1350 grams). Improvements in mean airway pressures began sometime between 2 and 6 hours in middle-size babies. These improvements persisted for at least 7 days.
Efficacy and impressive safety profile of EXOSURF® Neonatal™ confirmed in continued widespread use

In North American controlled clinical trials, more than 2600 premature infants received EXOSURF Neonatal. Under the year-long Treatment IND, over 11,400 infants received EXOSURF Neonatal. In the six months following its release for marketing, EXOSURF Neonatal has been given to 10,000 infants in more than 750 hospitals.

There are no known infectious or immunologic risks associated with EXOSURF Neonatal use. In controlled clinical trials, adverse events were comparable to those of placebo, with the exception of apnea and pulmonary bleeding. Infants receiving EXOSURF Neonatal required less ventilatory support, possibly contributing to an increased incidence of apnea. Pulmonary bleeding occurred in 1% of control infants and 2% of treated infants in controlled trials. In the treatment IND, pulmonary bleeding was reported in 4% and mucous plugging at a rate of 3/1000. Pulmonary bleeding appears to be preventable with early diagnosis and appropriate treatment of patent ductus arteriosus.

One-year follow-up evaluated developmental outcomes

Double-blind 1-year follow-up of more than 1450 infants enrolled in randomized trials showed that mental and motor scores appeared to be higher in tiny infants (<750 grams) as well as middle-size infants (750-1249 grams) who received Exosurf Neonatal.

Economic data analysis showed cost savings

Three separate studies evaluated the economic impact of a single prophylactic dose of EXOSURF Neonatal, two-dose rescue treatment in 700- to 1350-gram infants, and two-dose rescue treatment during the neonatal period in infants weighing over 1350 grams. Results indicate that both prophylactic treatment and rescue treatment are cost-effective. Mean hospital charges were $6451 less for large infants receiving two-dose rescue treatment versus air in the first 28 days of life.

As easy to use as it is effective

- **Easy to store and use**  EXOSURF Neonatal may be stored at room temperature. Reconstituted suspension may be maintained refrigerated or at room temperature for up to 12 hours. Key items needed for administration are supplied in one kit.

- **Easy to administer**  Each EXOSURF Neonatal dose is administered in two 2.5-mL/kg half-doses without interrupting mechanical ventilation.

- **Easy on infant**  To assist the distribution of EXOSURF Neonatal in the lungs, the infant is simply turned from midline position to the right after the first half-dose, and from midline position to the left after the second half-dose.

References:

PROPHYLACTIC TREATMENT 

1. Prophylactic treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see Contraindications). 

2. Prophylactic treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity and 

3. Rescue treatment of infants who have developed RDS. 

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf nebulizer.
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Deviations in Function of Mechanical Ventilators during Hyperbaric Compression

Paul B Blach BA RRT, David A Desautels MPA RRT, and T James Gallagher MD

BACKGROUND: Because little is known of the effects of increased pressure on some mechanical ventilators, we studied the effects of a hyperbaric environment on the function of 19 mechanical ventilators. 13 of which had not previously been studied. METHODS & MATERIALS: Tests were performed on each of the following 19 ventilators: Bio-Med IC-2A, Bio-Med ET-3, Bio-Med MVP-10, and Bio-Med P-7; Babybird, IMVbird, and Urgencybird; Oxylog: Antovent 2000; Ohmeda Logic 07; pneuPac Model 2 and pneuPac PNS 106; Omni-Vent HBC; Hydraulic Emerson; Monaghan 225; Oxford (Penlon Ltd); Bird Mark 10 and Mark 14; and Bennett PK-2. Each ventilator was placed inside a hyperbaric chamber and adjusted to a rate of approximately 10 breaths/min, tidal volume of 1000 mL, and an inspiratory time of 1-2 s. Chamber pressure was then increased and output of the ventilator settings measured. The ventilators were grouped for evaluation into three functional groups: pneumatic time-cycled, pneumatic pressure-cycled, and volume-cycled (piston or bellows). RESULTS: Function of the ventilators was consistent within each group, with some minor exceptions; however, function varied between groups. Under the conditions of our study, the Oxford was the only currently available machine able to maintain rate, tidal volume, and inspiratory time under hyperbaric compression. CONCLUSIONS: The choice of a mechanical ventilator for use in a hyperbaric environment should be made carefully. Ventilator function may deviate from set levels during compression. A mechanical ventilator specifically developed for the hyperbaric environment is needed. (Respir Care 1991;36:803-814.)

Background

The use of hyperbaric chambers has grown rapidly in recent years worldwide. This growth has led to an expanding list of conditions and types of patients treated, many of whom are critically ill and may require mechanical ventilation. Although some hyperbaric chambers come equipped with an integral mechanical ventilator (for example, the Sechrist 2500B), many chambers do not. When a chamber is not equipped with a ventilator, hyperbaric medical personnel must decide what type to use in the chamber. Typical choices include small, portable, pneumatic ventilators, which, when needed, can be placed into the chamber with the patient. Proper care to the ventilator-dependent patient during compression requires an appropriate mechanical ventilator; in particular, alveolar ventilation must be maintained reliably throughout the course of hyperbaric therapy. Hypoventilation may cause respiratory acidemia and, during compression, predispose the patient to an increased risk of grand mal seizures and central nervous system damage from oxygen toxicity. 11 Despite the serious nature of such consequences, only a small number of ven-
tilators that meet the unique requirements of a hyperbaric environment have been studied during compression, and nearly all of these have exhibited alterations in function. These studies, though helpful in alerting clinicians to the occurrence of ventilator deviations during hyperbaric therapy, leave numerous ventilators untested and important questions unanswered: Does high pressure affect all mechanical ventilators or are certain deviations characteristic of particular types of ventilators? Furthermore, the cause or causes of the deviations have not, to date, been adequately explained. In an effort to answer some of these questions and find a suitable hyperbaric ventilator, we evaluated 19 different mechanical ventilators (Table 1) under conditions of varying ambient pressure.

Methods and Materials

We evaluated 19 ventilators in a multipurpose hyperbaric chamber (Vacudyne®). Either ventilators were provided by the manufacturers specifically for the study or ventilators available in our institution were tested and calibrated if necessary immediately before being entered into the study. Patient simulation was provided by a lung analog: test-lung compliance (Cₜₑ) and test-lung resistance (Rₜₑ) were set and verified by direct measurement: 0.045 L/cm H₂O [0.46 L/kPa] and 7.5 cm H₂O · s · L⁻¹ [0.068 kPa

Table 1. Ventilators Tested in the Hyperbaric Environment

<table>
<thead>
<tr>
<th>Ventilator Model</th>
<th>Manufacturer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time-Cycled</strong></td>
<td></td>
</tr>
<tr>
<td>Bio-Med ET-3*</td>
<td>Bio-Med Devices</td>
</tr>
<tr>
<td>Bio-Med MVP-10*</td>
<td>Bio-Med Devices</td>
</tr>
<tr>
<td>Bio-Med P-7*</td>
<td>Bio-Med Devices</td>
</tr>
<tr>
<td>Babybird</td>
<td>Bird Products Corp</td>
</tr>
<tr>
<td>IMVbird</td>
<td>Bird Products Corp</td>
</tr>
<tr>
<td>Urgencybird</td>
<td>Bird Products Corp</td>
</tr>
<tr>
<td>Oxylog*</td>
<td>Dräger Werk AG</td>
</tr>
<tr>
<td>Autovent 2000*</td>
<td>Life Support Products</td>
</tr>
<tr>
<td>Logic 07*</td>
<td>Ohmeda</td>
</tr>
<tr>
<td>pneuPAC Model 2*</td>
<td>pneuPAC Ltd</td>
</tr>
<tr>
<td>pneuPAC PNS 106*</td>
<td>pneuPAC Ltd</td>
</tr>
<tr>
<td>Omni-Vent HBC*</td>
<td>Stein-Gates Medical</td>
</tr>
</tbody>
</table>

**Volume-Cycled**

Hydraulic Emerson        | JH Emerson Co         |
Monaghan 225*            | Monaghan Medical Corp |
Oxford®                  | Penlon Ltd            |

**Pressure-Cycled**

Mark 10                  | Bird Products Corp    |
Mark 14                  | Bird Products Corp    |
PR-2                     | Puritan-Bennett Corp  |

*Indicates ventilators provided by manufacturers specifically for the study. Other ventilators were hospital equipment that was tested and calibrated as necessary before being studied.

The values were maintained throughout the study. A pneumotachograph, the usual means of measuring flow and volume, was not practical for use in this study because the continuously changing gas density during compression would require recalibration of the pneumotachograph with each depth change. Such a time-consuming procedure would extend ‘bottom-time’ (ie, time subjected to increased pressure), which places the observer at increased risk for decompression illness; therefore, an alternative method for measuring tidal volume (Vₜₑ) was devised. The first step was to determine the linearity of the pressure-volume (compliance) setting selected on the test lung. With a 3-L calibration syringe, the volume of the test lung was increased progressively from 100 to 1,000 mL in 100-mL increments; with
each increase in volume, the alveolar pressure \( (P_A) \) in the test lung was measured at the intrapulmonary port and recorded on a calibrated strip-chart recorder. Pressure and volume data were plotted and a linear regression performed \( (r^2 = 0.998) \). Testing at both 30 feet of sea water (fsw), which is equal to 1.9 atmospheres absolute (ATA), and 60 fsw (2.8 ATA) provided essentially identical results.

The linear response of the \( C_{TL} \) and continuous recording of \( P_A \) within the test lung allows \( V_T \) to be determined from the mathematical relationship:

\[
V_T = \frac{(\Delta P_A)}{C_{TL}},
\]

where \( \Delta P_A \) is the difference between end-inspiratory \( P_A \) and end-expiratory \( P_A \) (Fig. 1, left).

The test lung, adjusted to the previously stated settings, was placed inside the hyperbaric chamber with the ventilator (Fig. 2) and an observer. Pressure and time were also determined from the tracing produced by the strip-chart recorder (Fig. 1, right), which was outside the chamber, via a penetration plug to the transducer. \( V_T \), determined as previously described, was verified visually at each depth by utilizing the \( V_T \) scale on the test lung. Transducer function was confirmed by comparing pressure readings from aneroid gauges on the test lung and ventilator to the strip-chart recording. Measurements were repeated at a compliance of 0.02 L/cm H\_2O [0.204 L/kPa]. Some resistive impedance was deemed necessary, but only one resistance setting was used because the response of factory-calibrated resistors during compression cannot be predicted accurately.

Throughout the study, ventilators were supplied with oxygen compressed at 50 psig [346.6 kPa] above ambient pressure within the hyperbaric chamber. Each ventilator was adjusted to a frequency \( (f) \) of approximately 10 breaths/min, \( V_T \) of 1,000 mL, and inspiratory time \( (t_i) \) of 1-2 seconds and was not altered during compression. In addition, we calculated an average inspiratory flowrate \( (\dot{V}_i) \) from \( V_T \) and \( t_i \) by the formula:

\[
\dot{V}_i = \frac{V_T}{t_i} \times 60.
\]

where flow rate is in L/min, \( V_T \) in L, and time in seconds.

![Fig. 1. Left: The strip-chart recording is marked for the pressure at end-inhalation \( (P_I) \), the pressure at end-expiration \( (P_E) \), and \( P_A \) computed by subtraction: \( \Delta P_A = P_I - P_E \). Tidal volume is determined by applying the mathematical relationship: \( V_T = P_A \times C_{TL} \). Some resistive impedance was deemed necessary, but only one resistance setting was used because the response of factory-calibrated resistors during compression cannot be predicted accurately.](image1)

![Fig. 2. Equipment configuration with a lung simulator to test ventilators under hyperbaric conditions.](image2)
by ventilating the chamber continuously with compressed air.

Results

To facilitate comparison, ventilators were placed in one of three groups based on ventilator design and cycling mechanism: pneumatic time-cycled (PTC), pneumatic pressure-cycled (PPC), and volume-cycled (VC) (piston or bellows) ventilators. Performance data of ventilators in each group were so similar that the data at each level of compression for each group were averaged and plotted (Figs. 3-7). The performance of all ventilators under each pressure with $C_{T}$ 0.045 L/cm H₂O [0.46 L/kPa] are tabulated in the Appendix (Tables 1-5). The results at the compliance level of 0.02 L/cm H₂O [0.204 L/kPa] were similar and are not reported.

Functions of two of the VC ventilators (Emerson and Oxford) were nearly constant at all levels of compression (Fig. 3, Appendix Tables 1-5). However, with the Monaghan 225, although $V_T$ remained constant, all other functions were progressively affected as compression increased, $f$ being 36%, $t_i$ and inspiratory time ($t_i$) 240% and 255%, respectively; and $V_t$ 44% of their control settings at maximal compression (165 fsw [6 ATA]) (Figs. 4-6, Appendix Tables 1-5).

Fig. 3. Volume with pneumatic time-cycled (circle), volume-cycled (square), and pneumatic pressure-cycled (triangle) ventilators as compression increases.

Fig. 4. Inspiratory time with pneumatic time-cycled (circle), volume-cycled (square), and pneumatic pressure-cycled (triangle) ventilators as compression increases.

Fig. 5. Expiratory time with pneumatic time-cycled (circle), volume-cycled (square), and pneumatic pressure-cycled (triangle) ventilators as compression increases.

Fig. 6. Rate with pneumatic time-cycled (circle), volume-cycled (square), and pneumatic pressure-cycled (triangle) ventilators as compression increases.
VENTILATOR FUNCTION DURING HYPERBARIC COMPRESSION

From 0 to 60 fsw (1 to 2.8 ATA), the PPC ventilators produced a consistent \( V_T \); but, at increased levels of compression, \( V_T \)s were lower than control settings (Fig. 3; Appendix Table 1). Rate was lower than its setting with the Bennett PR-2 and unaffected with the Bird Mark 10 and Mark 14 (Fig. 6, Appendix Table 4). Inspiratory time was higher than its control setting and flowrate was lower in each case. Expiratory time was decreased for the group, except in the PR-2, which increased to 182% (Fig. 4, Appendix Table 3).

Function of the PTC ventilators, as a group, was poorest: \( V_T \) was 14%; \( t_i \) and \( t_e \) 35% and 31%, respectively; and \( \dot{V}_l \) 43% of the control settings; \( f \) was increased in each case by an average of 326%, ranging from a low of 183% to a high of 651% (Figs. 3-7, Appendix Tables 1-4).

\( \dot{V}_l \) was decreased from its control setting as pressure increased for each ventilator studied, with two exceptions (Fig. 7, Appendix Table 5): No measurable deviation occurred at any level of compression with the Oxford, and a small increase occurred at 120 (4.6 ATA) and at 165 (6 ATA) fsw with the Hydraulic Emerson.

**Discussion**

A number of important questions remain unanswered concerning the function of mechanical ventilators during hyperbaric compression. For this reason, we studied 13 ventilators that have not been previously studied. In addition, to expand on earlier work, we re-evaluated 6 previously reported ventilators including: IMVbird, Urgencybird, pneuPAC Model 2, Hydraulic Emerson, Monaghan 225, and the Penlon Oxford.

It is likely that the continuous increase in gas density caused by compression is responsible for some of the deviations in ventilator function. Increased gas density, for instance, decreases flow through an orifice or needle valve as predicted by the equation for bulk gas flow:

\[
\text{flow} \propto \frac{1}{\text{gas density}}. \tag{3}
\]

Performance of many ventilators studied to date reflects the results of this effect. In one study of a PPC ventilator (Minutemen), \( t_i \), lengthened progressively with compression; increased gas density during compression may have prolonged the time required to achieve cycling pressure by decreasing inspiratory flowrate. Each of the three additional PPC ventilators we studied (Bird Mark 10, Bird Mark 14, and Bennett PR-2) functioned similarly. Another example is the Monaghan 225 (a VC ventilator); to deliver \( V_T \), gas passing through the inspiratory-flow needle valve is used to compress a bellows enclosed in a canister. The reduced gas flow during compression extended the time required to compress the bellows, which prolonged \( t_i \), as reported previously and reconfirmed in our study. With the exception of the Oxford and Hydraulic Emerson, each ventilator we tested exhibited a reduced \( \dot{V}_l \) during compression (Fig. 7, Appendix Table 5).

Several ventilators, including the pneuPAC (Model 2) and the Motivus (Type PV), have been reported to increase \( f \) progressively as compression increases and, to date, the factor responsible has not been clearly identified. In another study, \( V_T \) was reduced and \( f \) doubled with both the IMVbird and the Urgencybird. The investigators attempted
to restore the $V_T$ by completely opening the inspiratory flow control. They posited that opening the inspiratory flow control to its maximal point would cause the timing circuit to pressurize rapidly and, thus, increase rate. This seems unlikely because each of the 13 PTC ventilators included in our study increased rate during compression, regardless of the flow-control setting. Alterations in $t_i$, $t_e$, or both can alter ventilator rate. Our data reveal that rate increases observed with the PTC ventilators are the result of similar and progressive decreases in both $t_i$ and $t_e$ (Figs. 4 & 5, Appendix Tables 2 & 3). A nearly constant I.E. regardless of the level of compression, demonstrates the similarity of changes observed in $t_i$ and $t_e$.

In order to characterize ventilator function during hyperbaric compression according to type of ventilator, we classified the ventilators in our study by cycling mechanism, which was a successful strategy. For example, simultaneous deviations in $t_i$, $t_e$, and $f$ are apparently characteristic of PTC ventilators when functioning under conditions of changing ambient pressure. Because each of these variables is determined by the timing circuit within the PTC ventilator, it seems likely that hyperbaric compression affects some critical component or process essential to stable timing. In a typical pneumatic-timing circuit, gas under high pressure is metered through a precision needle valve (inspiratory-time control) and into a sequencing cartridge and an accompanying rigid vessel often referred to as a "timing chamber" (Fig. 8). Inspiratory time, which is defined as the interval required to fill the sequencing cartridge and timing chamber, depends on drive pressure exerted against the needle valve, size of the orifice within the needle valve, timing chamber volume, density of the source gas, and the compressibility of gas within the timing chamber. During the expiratory phase, gas that has accumulated within the timing chamber during inspiration meters out through a second needle valve (expiratory-time or rate control) (Fig. 9). Expiratory time ($t_e$), defined as the interval required to empty the timing chamber, depends on essentially the same factors as the inspiratory interval. In this study, drive pressure, orifice sizes, and timing-chamber volume were constant throughout compression and therefore did not affect timing; however, gas density, which increases with compression, decreased flow through the needle valves. Theoretically, this should prolong each timing interval, but our data show the opposite.

![Fig. 8. Inspiratory phase within a representative pneumatic timing circuit. Inspiration begins when the pressure inside the timing chamber and sequencing cartridge drops low enough to permit the sequencing cartridge to open. High pressure flows through the cartridge (A) and provides gas flow for mechanical inspiration (B). Simultaneously, high-pressure gas opens and flows through the expiratory isolation valve (C), meters through the inspiratory time control, and fills the sequencing cartridge (D) and the timing chamber (E). Gas is prevented from exiting the timing chamber and cartridge by a flow of high-pressure gas (F) that closes the inspiratory isolation valve. Throughout inspiration, a small amount of gas escapes into the room (G) via the discharge orifice.](image-url)

The only remaining factor is the compressibility of the gas within the timing chamber. The compressibility of a gas ($G_c$) with a volume ($V$) and a
Fig. 9. Expiratory phase within a representative pneumatic timing circuit. Expiration begins when the timing chamber and the sequencing cartridge are completely pressurized (filled) and the cartridge closes, terminating inspiratory gas flow. High-pressure gas, trapped inside the timing circuit during inspiration, closes the expiratory isolation valve and begins to meter out of the sequencing cartridge (A), timing chamber (B), and through the expiratory-time control. The high pressure retained within the timing circuit allows the exiting gas to open and flow through the inspiratory isolation valve (C). Gas exits the ventilator into the room (D) via the discharge orifice.

Pressure (P) is defined as the volume change per unit of pressure, mathematically:

\[
G_c = \frac{\Delta V}{\Delta P}. \tag{4}
\]

Utilizing Boyle's law and assuming isothermal conditions, the equation may be derived:

\[
G_c = \frac{V_c}{P_{atm}}, \tag{5}
\]

\(V_c\) = timing chamber volume and \(P_{atm}\) = atmospheric pressure.

According to this derived equation, when \(V_c\) remains constant, \(G_c\) is inversely proportional to \(P_{atm}\) and therefore decreases with progressive hyperbaric compression. This affects the pneumatic timing circuit because any specific volume of gas that meters into or out of the timing chamber produces a greater pressure change; thus, the chamber fills or empties in less time. The changes in timing observed in previous studies as well as ours are not of the magnitude predicted by the derived equation. This discrepancy is partially explained by the counteracting increase in gas density during compression, which reduces gas flow and prolongs or emptying of the timing circuit. Additional data conflicting with our hypothesis, that two PTC ventilators (modified Bird Mark 2 and IMVbird) decreased rate during compression, require an explanation. Within a pneumatic timing circuit, any increase or decrease in the drive pressure affects timing. It is likely that the observed changes in rate, in this instance, resulted from an alteration in drive pressure, and not from some hitherto unexplained effect of hyperbaric compression. Future studies should shed more light on the interaction between variations in atmospheric pressure, compressibility, and gas density, as they relate to mechanical ventilation.

Previous findings, as well as ours, indicate that PTC ventilators cannot be considered practical for use during hyperbaric therapy. Each ventilator in our study exhibited large and progressive deviations from each control setting as ambient pressure increased. Of most concern, \(V_T\) was reduced, on the average, by 14% of its control setting at the point of maximal compression (165 fsw [6 ATA]) (Fig. 3, Appendix Table 1). Although \(V_T\) and other parameters might be restored by an on-board attendant, such readjustments are not always successful, particularly at depths greater than 60 fsw (2.8...
ATA). Moreover, the task of having to constantly monitor and readjust so many ventilator variables reduces the time available for monitoring other patient parameters and maintaining other life-support equipment such as intravenous and arterial lines.

PPC ventilators are also poor choices for hyperbaric therapy; even at normobaric pressures, VT delivery depends directly on lung compliance and airways resistance (R_{aw}). A sudden, marked change in either or both can result in a potentially dangerous variation in VT. During the course of hyperbaric therapy, even if physiologic variables remain constant, some reduction in VT is inevitable because the increased gas density increases R_{aw} (Fig. 3, Part 1: Appendix Table 1). Furthermore, each PPC ventilator significantly prolonged the inspiratory interval during compression. Gas trapping, intrinsic PEEP, and barotrauma are all possible if a prolonged t_i is coupled with a reduced t_e, as observed with both the Bird Mark 10 and Mark 14 PPC ventilators. Nevertheless, a PPC ventilator can be safely used in a multiplace hyperbaric chamber, provided the attendant frequently monitors VT and the ventilator has sufficient inspiratory flow capacity to permit restoration of the I:E to its original setting.

The VC ventilators had the best overall hyperbaric performance. However, drawbacks exist to each of the three VC ventilators studied. They are all relatively large and all require modification to meet the criteria for suitability in a hyperbaric environment (Table 2). The Hydraulic Emerson delivers only controlled ventilation, is lubricated with a potentially flammable substance, and is not now commercially available. The Monaghan 225 experiences reduced f and prolonged t_i (by nearly threefold) (Appendix Table 2). These changes can be partially restored by adjusting ventilator settings and modifying the ventilator: The Monaghan may be modified to operate at a higher drive pressure, a scavenger system can be applied to the exhalation valve, and a separate system can be used to power the ventilator with air to allow safer and more effective ventilation in the hyperbaric environment. Even with these modifications, an attendant must adjust the ventilator with each depth change during compression. This renders the Monaghan unacceptable for use in monoplace hyperbaric chambers. The Oxford Penlon ventilator, constructed to deliver controlled ventilation, can be modified to deliver intermittent mandatory ventilation, positive end-expiratory pressure, and spontaneous ventilation.11

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Applicability to Hyperbaric Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple ventilatory modes</td>
<td>To provide most ventilatory modes deemed necessary to manage critically ill patients (NC, IMV, CPAP, PS)</td>
</tr>
<tr>
<td>Pneumatically driven</td>
<td>To avoid the fire hazard associated with electrical apparatus in hyperbaric environments</td>
</tr>
<tr>
<td>Stable operation</td>
<td>To reliably maintain all ventilatory parameters at baseline (1 atmosphere) setting, regardless of the level of compression</td>
</tr>
<tr>
<td>Compact</td>
<td>To conserve limited chamber space</td>
</tr>
<tr>
<td>Scavenged</td>
<td>To provide for simple collection and removal of all used pneumatic and ventilatory gases (to prevent the buildup of O_2 [fire hazard] and CO, [ventilatory stimulant])</td>
</tr>
<tr>
<td>Gas specific</td>
<td>To allow the operator to instantly switch ventilatory gases (air-oxygen, or 50-50 nitrox as dictated by specific treatment schedules)—Changing gases should not alter ventilator operation</td>
</tr>
<tr>
<td>Portable</td>
<td>To allow transport of patients to and from the chamber for therapy—A ventilator capable of both transport and hyperbaric operation offers many advantages</td>
</tr>
</tbody>
</table>

Based on the conditions utilized in our study and others,12 the Oxford appears to be the best overall choice of the ventilators we studied and is suitable for a monoplace hyperbaric chamber, provided there is sufficient space. The Oxford is not without
critics, however; during hyperbaric compression to 165 fsw (6 ATA), the Oxford has been reported to suffer progressive increases in rate and alterations in I:E as a function of increasing $R_{aw}$.\textsuperscript{12}

We evaluated no ventilators that are integral to hyperbaric chambers. However, in a 1988 study, the Sechrist 500A (integral to the 2500B hyperbaric chamber) altered $V_T$, $t_i$, and $f$ during hyperbaric compression.\textsuperscript{4}

**Conclusion**

Ventilators suitable for use in a hyperbaric chamber should possess certain characteristics (Table 2). In addition, the ventilator should be easy to use and have uncomplicated circuitry. Most importantly, ventilator function should not be markedly affected by changes in ambient pressure; this is critical when ventilators are used in monoplace chambers.

Currently (1991), no ventilator available meets all the specifications for suitability in a hyperbaric environment. From our discussions with representatives of industry, we conclude that demand for such a ventilator is not high. The demand probably does not justify the expenses involved in research, development, and marketing. Until the dilemma of demand versus cost is resolved and a ventilator specifically designed for hyperbaric use is available, we advise caution in selecting ventilators for hyperbaric use. Furthermore, we must continue to rely upon skilled attendants to diligently monitor ventilators and patients throughout the course of hyperbaric therapy and to restore ventilator parameters as they are altered by compression. Additional studies aimed at understanding how compression affects ventilator function should help us accomplish this task and provide a better understanding of ventilators in general.

**ACKNOWLEDGMENTS**

We thank Richard J Melker MD PhD for his help in obtaining many of the ventilators and Lynn Dirk for her editorial assistance.

**PRODUCT SOURCES**

**Multiplace hyperbaric chamber:**
Vacadyne, Chicago Heights IL

**Pressure-cycled ventilators:**
Bird Mark 10 and Mark 14, Bird Products Corp, Palm Springs CA
PR-2, Puritan-Bennett Corp. Overland Park KS

**Time-cycled ventilators:**
Babybird, IMVbird, Urgencybird, Bird Products Corp, Palm Springs CA
Oxylog, Dräger Werk AG, Lubeck, Germany
Autovent 2000, Life Support Products, Irvine CA
Logic 07, Ohmeda, Madison WI
pneuPAC Models 2 and 106, pneuPAC Ltd, Luton-Beds, UK
Omni-Vent HBC, Stein Gate, Medical, Atchinson KS

**Volume-cycled ventilators:**
Hydraulic Emerson, JH Emerson Co, Cambridge MA
Monaghan 225, Monaghan Medical Corp, Plattsburgh NY
Oxford, Penlon Ltd, Abingdon UK

**Test lung:**
TTL, Michigan Instruments, Grand Rapids MI

**Calibration syringe:**
3-L syringe, Warren E Collins, Braintree MA

**Calibrated strip-chart recorder:**
Pneumogard, Novametrix, Wallingford CT

**Transducer:**
Bentley, Bentley Laboratories, Irvine CA

**REFERENCES**

## APPENDIX

### Tables 1-5

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Performance of Ventilators under Hyperbaric Conditions—Effect on Tidal Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depth in Feet of Sea Water</strong></td>
<td><strong>(absolute atmospheres)</strong></td>
</tr>
<tr>
<td><strong>(1 ATA)</strong></td>
<td><strong>(1.2 ATA)</strong></td>
</tr>
<tr>
<td><strong>Ventilator</strong></td>
<td><strong>Tidal Volume in ml.</strong></td>
</tr>
<tr>
<td>Bro-Med IC-2A</td>
<td>951 (12)</td>
</tr>
<tr>
<td>Bio-Med FT-3</td>
<td>973 (13)</td>
</tr>
<tr>
<td>Bio-Med MVP-10</td>
<td>906 (0)</td>
</tr>
<tr>
<td>Bio-Med P-7</td>
<td>1075 (6)</td>
</tr>
<tr>
<td>Rollybord</td>
<td>1036 (10)</td>
</tr>
<tr>
<td>IMV/Mark</td>
<td>1008 (20)</td>
</tr>
<tr>
<td>Urgencybord</td>
<td>932 (10)</td>
</tr>
<tr>
<td>Oxilog</td>
<td>891 (14)</td>
</tr>
<tr>
<td>Automed 2000</td>
<td>985 (4)</td>
</tr>
<tr>
<td>Logiq 07</td>
<td>957 (35)</td>
</tr>
<tr>
<td>pneumPAC 2</td>
<td>1072 (0)</td>
</tr>
<tr>
<td>pneumPAC 106</td>
<td>1016 (6)</td>
</tr>
<tr>
<td>Otto-Vent</td>
<td>954 (9)</td>
</tr>
<tr>
<td>Mean</td>
<td>962 (48)</td>
</tr>
</tbody>
</table>

### Time-Cycled

- **Bro-Med IC-2A**: Mean: 951, SD: 12
- **Bio-Med FT-3**: Mean: 973, SD: 13
- **Bio-Med MVP-10**: Mean: 906, SD: 0
- **Bio-Med P-7**: Mean: 1075, SD: 6
- **Rollybord**: Mean: 1036, SD: 10
- **IMV/Mark**: Mean: 1008, SD: 20
- **Urgencybord**: Mean: 932, SD: 10
- **Oxilog**: Mean: 891, SD: 14
- **Automed 2000**: Mean: 985, SD: 4
- **Logiq 07**: Mean: 957, SD: 35
- **pneumPAC 2**: Mean: 1072, SD: 0
- **pneumPAC 106**: Mean: 1016, SD: 6
- **Otto-Vent**: Mean: 954, SD: 9
- **Mean**: Mean: 962, SD: 48

### Volume-Cycled

- **Emerson**: Mean: 990, SD: 0
- **Monograph**: Mean: 924, SD: 10
- **Oxford Fenelon**: Mean: 1013, SD: 9
- **Mean**: Mean: 976, SD: 46

### Pressure-Cycled

- **Bird Mark 10**: Mean: 990, SD: 0
- **Bird Mark 14**: Mean: 1022, SD: 9
- **Bennett PR-2**: Mean: 977, SD: 15
- **Mean**: Mean: 990, SD: 23

**RESPIRATORY CARE • AUGUST ’91 Vol 36 No 8**
### Table 2: Performance of Ventilators under Hypobaric Conditions—Effect on Inspiratory Time

<table>
<thead>
<tr>
<th>Time-Cycle</th>
<th>0 (1 ATA)</th>
<th>15 (0.15 ATA)</th>
<th>45 (0.45 ATA)</th>
<th>60 (0.6 ATA)</th>
<th>120 (0.2 ATA)</th>
<th>165 (0.165 ATA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emerson</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.96 (0.0)</td>
</tr>
<tr>
<td>Stryker</td>
<td>0.98 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
</tr>
<tr>
<td>Portex</td>
<td>0.97 (0.0)</td>
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</table>

### Table 3: Performance of Ventilators under Hypobaric Conditions—Effect on Expiratory Time

<table>
<thead>
<tr>
<th>Time-Cycle</th>
<th>0 (1 ATA)</th>
<th>15 (0.15 ATA)</th>
<th>45 (0.45 ATA)</th>
<th>60 (0.6 ATA)</th>
<th>120 (0.2 ATA)</th>
<th>165 (0.165 ATA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emerson</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.96 (0.0)</td>
</tr>
<tr>
<td>Stryker</td>
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<td>0.98 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
</tr>
<tr>
<td>Portex</td>
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<td>0.96 (0.0)</td>
<td>0.96 (0.0)</td>
<td>0.95 (0.0)</td>
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### Tables 2 and 3 continued

### Table 4: Performance of Ventilators under Hypobaric Conditions—Effect on Volume

<table>
<thead>
<tr>
<th>Time-Cycle</th>
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<th>45 (0.45 ATA)</th>
<th>60 (0.6 ATA)</th>
<th>120 (0.2 ATA)</th>
<th>165 (0.165 ATA)</th>
</tr>
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<tbody>
<tr>
<td>Emerson</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>1.00 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.96 (0.0)</td>
</tr>
<tr>
<td>Stryker</td>
<td>0.98 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.98 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
<td>0.97 (0.0)</td>
</tr>
<tr>
<td>Portex</td>
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<td>0.96 (0.0)</td>
<td>0.96 (0.0)</td>
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### Table 4: Performance of Ventilators under Hypobaric Conditions— Effect on Rate

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<th>Depth in feet of Sea Water (absolute atmospheres)</th>
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<th>45</th>
<th>60</th>
<th>120</th>
<th>165</th>
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<td>(1 ATA)</td>
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<td></td>
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<tr>
<td>(1.4 ATA)</td>
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<td></td>
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<td>(1.9 ATA)</td>
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<td></td>
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<td>(2.4 ATA)</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>(2.8 ATA)</td>
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<td></td>
<td></td>
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<td></td>
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<tr>
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<td></td>
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<tr>
<td>(6.6 ATA)</td>
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<td></td>
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</table>

<table>
<thead>
<tr>
<th>Ventilator</th>
<th>Rate in Breaths/Minute Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time-Cycled</td>
<td></td>
</tr>
<tr>
<td>Biosol IC-CA</td>
<td>10.4 (0.2)</td>
</tr>
<tr>
<td>Biosol ET-3</td>
<td>8.60 (0.1)</td>
</tr>
<tr>
<td>Biosol MVP 10</td>
<td>7.98 (0.09)</td>
</tr>
<tr>
<td>Biosol P-7</td>
<td>11.3 (0.2)</td>
</tr>
<tr>
<td>Biovent P</td>
<td>10.8 (0.2)</td>
</tr>
<tr>
<td>Biovent P</td>
<td>10.4 (0.1)</td>
</tr>
<tr>
<td>Urgency vent</td>
<td>10.0 (0.1)</td>
</tr>
<tr>
<td>Oxygen</td>
<td>9.70 (0.05)</td>
</tr>
<tr>
<td>Autovent 2000</td>
<td>9.30 (0.12)</td>
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<tr>
<td>Logic O7</td>
<td>8.40 (0.1)</td>
</tr>
<tr>
<td>pneuPAC 2</td>
<td>11.9 (0.09)</td>
</tr>
<tr>
<td>pneuPAC 106</td>
<td>10.8 (0.1)</td>
</tr>
<tr>
<td>Omnis Vent</td>
<td>10.2 (0.03)</td>
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<tr>
<td>Mean</td>
<td>10.18 (1.4)</td>
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<tr>
<td>Volume-Cycled</td>
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</tr>
<tr>
<td>Emerson</td>
<td>10.2 (0.05)</td>
</tr>
<tr>
<td>Monaghan</td>
<td>11.4 (0.1)</td>
</tr>
<tr>
<td>Oxford Penlon</td>
<td>9.92 (0.18)</td>
</tr>
<tr>
<td>Mean</td>
<td>10.15 (0.79)</td>
</tr>
<tr>
<td>Pressure-Cycled</td>
<td></td>
</tr>
<tr>
<td>Bird Mark 10</td>
<td>10.1 (0.1)</td>
</tr>
<tr>
<td>Bird Mark 14</td>
<td>10.6 (0.1)</td>
</tr>
<tr>
<td>Bennett P-8</td>
<td>10.9 (0.01)</td>
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<tr>
<td>Mean</td>
<td>10.2 (0.36)</td>
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### Table 5: Performance of Ventilators under Hypobaric Conditions— Effect on Inspiratory Flowrate

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<th>30</th>
<th>45</th>
<th>60</th>
<th>120</th>
<th>165</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1 ATA)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>(1.4 ATA)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1.9 ATA)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2.4 ATA)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2.8 ATA)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4.6 ATA)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(6.6 ATA)</td>
<td>1</td>
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<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Ventilator</th>
<th>Inspiratory Flowrate at L/min Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time-Cycled</td>
<td></td>
</tr>
<tr>
<td>Biosol IC-CA</td>
<td>33.1 (0.7)</td>
</tr>
<tr>
<td>Biosol ET-3</td>
<td>14.3 (1.2)</td>
</tr>
<tr>
<td>Biosol MVP 10</td>
<td>21.5 (0.5)</td>
</tr>
<tr>
<td>Biosol P-7</td>
<td>40.6 (1.2)</td>
</tr>
<tr>
<td>Biovent P</td>
<td>30.3 (0.7)</td>
</tr>
<tr>
<td>Biovent P</td>
<td>45.7 (1.7)</td>
</tr>
<tr>
<td>Urgency vent</td>
<td>29.2 (0.6)</td>
</tr>
<tr>
<td>Oxygen</td>
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</tr>
<tr>
<td>Autovent 2000</td>
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</tr>
<tr>
<td>Logic O7</td>
<td>22.2 (1.6)</td>
</tr>
<tr>
<td>pneuPAC 2</td>
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</tr>
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<td>pneuPAC 106</td>
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</tr>
<tr>
<td>Omnis Vent</td>
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</tr>
<tr>
<td>Mean</td>
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</tr>
<tr>
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<tr>
<td>Monaghan</td>
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</tr>
<tr>
<td>Pressure-Cycled</td>
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<td>Bird Mark 10</td>
<td>37.4 (0.8)</td>
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<tr>
<td>Bird Mark 14</td>
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<tr>
<td>Bennett P-8</td>
<td>26.9 (0.7)</td>
</tr>
<tr>
<td>Mean</td>
<td>33.9 (0.6)</td>
</tr>
</tbody>
</table>
Work of Breathing during CPAP and PSV Imposed by the New Generation Mechanical Ventilators: A Lung Model Study

Christopher Hirsch BS RRT, Robert M Kacmarek PhD RRT, and Kevin Stanek BS

BACKGROUND: Numerous mechanical ventilators are presently available—some have never been evaluated for imposed work of breathing (WOBi), and some have not been evaluated for WOBi subsequent to being upgraded by the manufacturer. In this study, we evaluated the WOBi of a free-standing CPAP system at 0 and 10 cm H2O CPAP and of 9 mechanical ventilators at 0 cm H2O PSV, 0 cm H2O CPAP; 0 cm H2O PSV, 10 cm H2O CPAP; 10 cm H2O PSV, 0 cm H2O CPAP; and 10 cm H2O PSV, 10 cm H2O CPAP. METHODS: All evaluations were performed using a 2-chamber lung model powered by a Bear 5 ventilator set at VT 300 mL, Vmax 20 L/min; VT 400 mL, Vmax 40 L/min; and VT 500 mL, Vmax 60 L/min. RESULTS: Minimal WOBi was measured at 0 cm H2O PSV, 0 cm H2O CPAP; 10 cm H2O PSV, 0 cm H2O CPAP; and 10 cm H2O PSV, 10 cm H2O CPAP. Markedly elevated WOBi was measured (with most systems) at 0 cm H2O PSV, 10 cm H2O CPAP. All demand systems produced low levels of PSV (1.5-5 cm H2O), even when the PSV level was set at 0 cm H2O. CONCLUSIONS: If properly maintained, most ventilator demand-valve systems impose minimal WOB that can be essentially eliminated by using 10 cm H2O PSV. The addition of CPAP increases WOBi, as does the use of a bubble-through humidifier. The free-standing CPAP system outperformed most ventilator demand-valve systems when a PEEP valve with minimal flow resistance was used and appropriate continuous flows were set. (Respir Care 1991;36:815-828.)

Background

The work imposed by mechanical ventilators during spontaneous breathing has been the subject of many reported studies. The findings of these studies have helped to shape protocols used for weaning patients from ventilatory support, and have encouraged the use of pressure support ventilation (PSV) as an adjunct to spontaneous ventilation to counteract the work imposed by demand systems and endotracheal tubes. The results of these studies have also encouraged manufacturers to modify demand systems by improving sensitivity, decreasing delay time, and increasing flow capabilities. These modifications and the use of PSV have reduced the work of breathing imposed (WOBi) by demand systems. Manufacturers have also introduced flow-triggering during continuous positive airway pressure (CPAP) (Puritan-Bennett 7200a) and the ability to vary the time required for peak flow requirements to be met during PSV (IRIS ventilator). The effects of these modifications on WOBi have not been evaluated. Also, the effect on WOBi of bubble-through versus passover humidification (during demand-flow CPAP) has not been evaluated.

Using a standard lung model, we determined WOBi and maximum inspiratory subbaseline press—
Abbreviations Used in This Paper

CPAP — Continuous positive-airway pressure
EP — End-inspiratory pressure
PEEP — Positive end-expiratory pressure
P_{max} — Maximum subatmospheric pressure developed during inspiration
PSV — Pressure support ventilation
t_i — Inspiratory time
V_{max} — Peak system flowrate
V_T — Tidal volume
WOB_i — Imposed work of breathing

WOB during CPAP and PSV using nine ventilators: Bird 6400ST, Hamilton Veolar, Siemens 900C, Puritan-Bennett 7200a, Intermed Bear 5, Intermed Bear 3, PPG Industries IRISA, Newport E100i, and Newport Breeze; and during CPAP using a free-standing CPAP system.

Methods

All comparisons were performed utilizing the Michigan Industries' double-chamber test lung. The setup of the lung model (Fig. 1) is similar to that used by Katz et al. One chamber of the test lung (driving chamber) was attached to and powered by a Bear 5 ventilator with a sine-wave flow pattern. The other chamber of the test lung (experimental chamber) was attached to the ventilator or CPAP system being evaluated. The two chambers were not physically connected; however, a small metal insert was incorporated that allowed the driving chamber to lift the experimental chamber. Thus, the establishment of positive pressure in the driving chamber created a subatmospheric pressure in the experimental chamber that triggered demand flow or a pressure-supported breath. Once flow was triggered, the experimental chamber could rise independent of the driving chamber. The compliances of both chambers were set at 45 mL/cm H_2O. A 9-mm-ID endotracheal tube was used to connect each ventilator (or CPAP system) to its respective chamber, and sufficient PEEP was applied to the driving chamber to prevent interchamber separation at end-expiration.

Suppliers are identified in the Product Sources section at the end of the text.

The circuit of the experimental ventilator was configured with a pressure tap and Hans Rudolph 3700 pneumotachograph in series between the distal end of the circuit Y and the proximal end of the endotracheal tube. The pressure tap led to a Validyne MP 45-32-871 ± 100 cm H_2O differential pressure transducer. Pressure and flow signals were measured and integrated by a DEC LSI 11/23 computer with an ADAC data-acquisition module and displayed as pressure-volume loops on a Tektronix 40006-1 graphics terminal. The raw data and the pressure-volume loops were computer analyzed. Variables measured included peak system flowrate (V_{max}); delivered tidal volume (V_T); inspiratory time (t_i); P_{max} (i.e., the subatmospheric pressure required to trigger demand flow or a pressure-supported breath); WOB_i during inspiration; and end inspiratory pressure (EP).

We defined inspiratory WOB as the area of the pressure-volume loop between the vertical line at the system's baseline pressure and that part of the pressure-volume curve (left of baseline) established by subbaseline pressure (Fig. 2). The work performed by the ventilator was not incorporated into the calculation of WOB_i; that is, the area to the right of baseline pressure during inspiration was disregarded during WOB_i determinations. This definition and determination of WOB_i have previously been described by Katz et al.

Prior to attaching the test lung to the ventilator or CPAP system being evaluated, the Bear 5 con-
nected to the driving component of the lung model was set at one of three \( V_t-V_{\text{max}} \) combinations (300 mL-20 L/min, 400 mL-40 L/min, or 500 mL-60 L/min), and actual \( V_t \) and \( V_{\text{max}} \) were verified at the location of the endotracheal tube attached to the experimental chamber. Each experimental and CPAP system was evaluated at all three lung-model \( V_t-V_{\text{max}} \) combinations.

All of the ventilators (with the exception of the Newport E100i and Newport Breeze) were evaluated in the following modes: (1) 0 cm H\(_2\)O PSV, 0 cm H\(_2\)O CPAP; (2) 0 cm H\(_2\)O PSV, 10 cm H\(_2\)O CPAP; (3) 10 cm H\(_2\)O PSV, 0 cm H\(_2\)O CPAP; and (4) 10 cm H\(_2\)O PSV, 10 cm H\(_2\)O CPAP. The two Newport ventilators were unable to provide PSV and therefore were evaluated only in the CPAP mode at 0 and 10 cm H\(_2\)O.

The free-standing continuous-flow CPAP system was evaluated only in the CPAP mode at 0 and 10 cm H\(_2\)O. At lung-model \( V_t-V_{\text{max}} \) combinations of 300 mL-20 L/min and 400 mL-40 L/min, continuous flow was maintained at 60 L/min; at 500 mL-60 L/min, continuous flow was set at 80 L/min. The CPAP system was set up as described by Kacmarek et al. A 5-L reservoir bag was used in the inspiratory limb distal to the system humidifier, and CPAP was established with a Vital Signs CPAP valve.

With the lung model set at \( V_t-V_{\text{max}} \) of 400 mL-40 L/min, we evaluated the effect of the 20-L/min base-flow options of the Bear 5 (continuous flow) and 7200a (flow-by) ventilators at both 0 and 10 cm H\(_2\)O CPAP. The flow sensitivity of the 7200a flow-by was set at 3 L/min during this evaluation.

The effect of altering the response time of the IRISA during PSV of 10 cm H\(_2\)O, with 0 and 10 cm H\(_2\)O CPAP, was also evaluated. Adjustment of response time alters the time required for peak flow requirements to be met. Two settings of response time, fastest and slowest, were evaluated.

With the lung model set at \( V_t-V_{\text{max}} \) of 400 mL-40 L/min, the effect of the tower in the Cascade II humidifier of the 7200a ventilator was evaluated at 0 and 10 cm H\(_2\)O CPAP. Measurements were made with and without the tower in place.

During our study, we used only ventilator circuits recommended by the manufacturers. Only passover humidification (Cascade I or II without tower) was used in our study, except during the 7200a ventilator evaluation. The working pressure of the Servo 900C was set at 70 cm H\(_2\)O during all evaluations. With the exception of the IRISA, which has a factory preset sensitivity of -0.7 cm H\(_2\)O, all ventilators were set at sensitivity of -1 cm H\(_2\)O. The highest continuous flows obtainable with the Newport E100i and Breeze, when configured as recommended by the manufacturer, were set. All ventilators either received preventive maintenance prior to the study or were on loan from the manufacturer; each ventilator was functioning at its optimal level. Data were obtained during 6 representative breaths for each ventilator and the CPAP system at each combination of settings, and means and standard deviations were determined.

**Results**

Data are presented in Tables 1-4 for WOB, \( P_{\text{max}}-\text{neg} \), \( V_{\text{max}} \), \( t_s \), \( V_t \), and EIP under all experimental conditions with the lung model set at \( V_t-V_{\text{max}} \) of 400 mL-40 L/min. Results when the lung
Table 1. Results of Bench Evaluation of Nine Ventilators and One Free-Standing, Continuous-Flow CPAP System at 0 cm H₂O PSV and 0 cm H₂O CPAP

<table>
<thead>
<tr>
<th>Ventilator</th>
<th>WOB&lt;sub&gt;i&lt;/sub&gt; (J/L)</th>
<th>P&lt;sub&gt;max-neg&lt;/sub&gt; cm H₂O</th>
<th>˙V&lt;sub&gt;max&lt;/sub&gt; L/min</th>
<th>t&lt;sub&gt;i&lt;/sub&gt; s</th>
<th>V&lt;sub&gt;T&lt;/sub&gt; mL</th>
<th>EIP cm H₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volar</td>
<td>0.009 (0.001)</td>
<td>-1.3 (0.2)</td>
<td>55 (0)</td>
<td>1.0 (0.0)</td>
<td>455 (3)</td>
<td>2.93 (0.84)</td>
</tr>
<tr>
<td>CPAP-S (CF 60)</td>
<td>0.012 (0.002)</td>
<td>-0.7 (0.1)</td>
<td>45 (1)</td>
<td>1.0 (0.0)</td>
<td>459 (4)</td>
<td>— —</td>
</tr>
<tr>
<td>7200a (CF 20)</td>
<td>0.022 (0.003)</td>
<td>-0.8 (0.2)</td>
<td>40 (0)</td>
<td>1.0 (0.0)</td>
<td>434 (3)</td>
<td>2.2 (0.43)</td>
</tr>
<tr>
<td>IRISA</td>
<td>0.025 (0.004)</td>
<td>-0.9 (0.2)</td>
<td>41 (1)</td>
<td>0.9 (0.0)</td>
<td>421 (4)</td>
<td>1.45 (0.34)</td>
</tr>
<tr>
<td>Bear 3</td>
<td>0.025 (0.001)</td>
<td>-2.1 (0.2)</td>
<td>55 (0)</td>
<td>0.8 (0.0)</td>
<td>458 (1)</td>
<td>1.85 (0.17)</td>
</tr>
<tr>
<td>7200a</td>
<td>0.032 (0.006)</td>
<td>-0.8 (0.2)</td>
<td>39 (1)</td>
<td>1.0 (0.0)</td>
<td>430 (1)</td>
<td>2.75 (0.5)</td>
</tr>
<tr>
<td>Bear 5 (CF 20)</td>
<td>0.035 (0.000)</td>
<td>-0.9 (0.1)</td>
<td>41 (0)</td>
<td>1.0 (0.0)</td>
<td>416 (1)</td>
<td>2.22 (0.35)</td>
</tr>
<tr>
<td>E100i (CF 48)</td>
<td>0.052 (0.002)</td>
<td>-1.6 (0.1)</td>
<td>39 (1)</td>
<td>0.9 (0.0)</td>
<td>400 (1)</td>
<td>— —</td>
</tr>
<tr>
<td>6400ST</td>
<td>0.054 (0.006)</td>
<td>-2.1 (0.3)</td>
<td>47 (1)</td>
<td>0.9 (0.0)</td>
<td>459 (1)</td>
<td>1.56 (0.21)</td>
</tr>
<tr>
<td>900C</td>
<td>0.063 (0.008)</td>
<td>-2.6 (0.1)</td>
<td>63 (1)</td>
<td>0.7 (0.0)</td>
<td>400 (3)</td>
<td>1.80 (0.13)</td>
</tr>
<tr>
<td>Bear 5</td>
<td>0.073 (0.012)</td>
<td>-2.8 (0.3)</td>
<td>55 (0)</td>
<td>1.0 (0.0)</td>
<td>410 (3)</td>
<td>2.42 (0.26)</td>
</tr>
<tr>
<td>Breeze (CF 53)</td>
<td>0.078 (0.009)</td>
<td>-1.2 (0.1)</td>
<td>39 (2)</td>
<td>1.0 (0.0)</td>
<td>408 (2)</td>
<td>— —</td>
</tr>
<tr>
<td>7200a (Tower)</td>
<td>0.093 (0.004)</td>
<td>-1.5 (0.2)</td>
<td>41 (0)</td>
<td>0.9 (0.0)</td>
<td>435 (3)</td>
<td>2.08 (0.43)</td>
</tr>
</tbody>
</table>

* Lung model peak flow set at 40 L/min, tidal volume at 400 mL.
* All values are mean (SD).
* CPAP-S is the free-standing, continuous-flow CPAP system.
* CF = continuous gas flow settings in L/min.
* II Cascade tower in place; passover humidification used with all other systems.

The model was set at V<sub>T</sub>- ˙V<sub>max</sub> of 300 mL-20 L/min and 500 mL-60 L/min reveal the same comparative values for each ventilator evaluated and across experimental conditions and are thus not presented. Trends of all data are reflected in Tables 1-4; ventilators are listed by levels of WOB<sub>i</sub>, those imposing the least WOB are listed first and those imposing the greatest last.

Figures 3-6 depict representative pressure-volume loops obtained during all combinations of CPAP and PSV with the lung model set at V<sub>T</sub>- ˙V<sub>max</sub> of 400 mL-40 L/min.

**Discussion**

Our results indicate ventilator-demand and continuous-flow systems function differently at 0 and 10 cm H₂O CPAP. These differences were also noted (to a lesser extent) when PSV of 10 cm H₂O was added to 0 and 10 cm H₂O CPAP. However, the addition of 10 cm H₂O PSV all but eliminated the WOB<sub>i</sub> of all ventilator demand systems—even during 10 cm H₂O CPAP. No single ventilator consistently outperformed all others, although the Volar functioned best in the CPAP modes without
Table 2. Results of Bench Evaluation of Nine Ventilators and One Free-Standing, Continuous-Flow CPAP System at 0 cm H₂O PSV and 10 cm H₂O CPAP

<table>
<thead>
<tr>
<th></th>
<th>WOB&lt;sub&gt;i&lt;/sub&gt;</th>
<th>P&lt;sub&gt;max-neg&lt;/sub&gt;</th>
<th>V&lt;sub&gt;max&lt;/sub&gt;</th>
<th>t&lt;sub&gt;i&lt;/sub&gt;</th>
<th>V&lt;sub&gt;T&lt;/sub&gt;</th>
<th>EIP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>J/L</td>
<td>cm H₂O</td>
<td>L/min</td>
<td>s</td>
<td>mL</td>
<td>cm H₂O</td>
</tr>
<tr>
<td>Veolar</td>
<td>0.066 (0.037)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-2.2 (0.7)</td>
<td>45 (1)</td>
<td>1.0 (0.1)</td>
<td>458 (2)</td>
<td>5.0 (1.16)</td>
</tr>
<tr>
<td>7200a (CF 20)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.078 (0.005)</td>
<td>-1.2 (0.1)</td>
<td>43 (0)</td>
<td>0.9 (0.0)</td>
<td>409 (2)</td>
<td>2.6 (0.49)</td>
</tr>
<tr>
<td>900C</td>
<td>0.091 (0.030)</td>
<td>-3.9 (0.2)</td>
<td>63 (3)</td>
<td>0.7 (0.0)</td>
<td>429 (1)</td>
<td>3.8 (1.47)</td>
</tr>
<tr>
<td>CPAP-S&lt;sup&gt;c&lt;/sup&gt; (CF 60)</td>
<td>0.107 (0.002)</td>
<td>-1.47 (0.1)</td>
<td>42 (0)</td>
<td>0.8 (0.0)</td>
<td>390 (1)</td>
<td>-</td>
</tr>
<tr>
<td>7200a</td>
<td>0.0114 (0.015)</td>
<td>-2.0 (0.3)</td>
<td>44 (1)</td>
<td>0.9 (0.0)</td>
<td>395 (1)</td>
<td>3.6 (1.05)</td>
</tr>
<tr>
<td>7200a (Tower iII)</td>
<td>0.194 (0.132)</td>
<td>-4.1 (0.2)</td>
<td>46 (3)</td>
<td>0.9 (0.1)</td>
<td>390 (4)</td>
<td>2.56 (0.21)</td>
</tr>
<tr>
<td>6400ST</td>
<td>0.256 (0.025)</td>
<td>-3.3 (0.2)</td>
<td>48 (0)</td>
<td>0.9 (0.0)</td>
<td>465 (2)</td>
<td>2.2 (0.24)</td>
</tr>
<tr>
<td>Bear 5</td>
<td>0.268 (0.119)</td>
<td>-2.5 (0.4)</td>
<td>44 (2)</td>
<td>0.9 (0.0)</td>
<td>428 (3)</td>
<td>3.4 (0.76)</td>
</tr>
<tr>
<td>Bear 3</td>
<td>0.286 (0.044)</td>
<td>-4.2 (0.2)</td>
<td>55 (0)</td>
<td>0.8 (0.0)</td>
<td>452 (1)</td>
<td>4.4 (0.21)</td>
</tr>
<tr>
<td>IRISA</td>
<td>0.291 (0.095)</td>
<td>-0.9 (0.4)</td>
<td>40 (1)</td>
<td>0.9 (0.0)</td>
<td>390 (2)</td>
<td>2.62 (0.67)</td>
</tr>
<tr>
<td>E100i (CF-48)</td>
<td>0.553 (0.005)</td>
<td>-2.4 (0.1)</td>
<td>39 (1)</td>
<td>1.0 (0.0)</td>
<td>390 (1)</td>
<td>-</td>
</tr>
<tr>
<td>Bear 5 (CF 20)</td>
<td>0.634 (0.024)</td>
<td>-3.0 (0.5)</td>
<td>47 (1)</td>
<td>1.0 (0.0)</td>
<td>424 (2)</td>
<td>3.4 (0.16)</td>
</tr>
<tr>
<td>Breeze (CF 53)</td>
<td>0.735 (0.008)</td>
<td>-2.38 (0.1)</td>
<td>40 (2)</td>
<td>1.0 (0.0)</td>
<td>412 (1)</td>
<td>-</td>
</tr>
</tbody>
</table>

<sup>a</sup> Lung model peak flow set at 40 L/min, tidal volume at 400 mL.
<sup>b</sup> All values are mean (SD).
<sup>c</sup> CF = continuous gas flow settings in L/min.
<sup>d</sup> CPAP-S is the free-standing, continuous-flow CPAP system.
<sup>e</sup> Cascade tower in place; passover humidification used with all other systems.

PSV (Tables 1 and 2), and the 7200a outperformed the other units at 10 cm H₂O PSV, 10 cm H₂O CPAP (Table 4). All ventilators essentially performed consistently at 10 cm H₂O PSV, 0 cm H₂O CPAP (Table 3). At 0 cm H₂O CPAP, the free-standing CPAP system outperformed all ventilators except the Veolar (Table 1); at 10 cm H₂O CPAP the CPAP system was outperformed by the Veolar, 7200a (Flow-by), and 900C (Table 2).

Summarized in Table 5 are the results of three other studies<sup>6,7,17</sup> that used the same lung model to evaluate WOB<sub>i</sub> for similar ventilators. Using the Katz lung model, Fiastro et al<sup>11</sup> measured WOB<sub>i</sub> of approximately 0.220 J/L with the 7200a at 40 L/min peak inspiratory flow and 0 cm H₂O CPAP. In a series of patients, Gibney et al<sup>18</sup> measured WOB<sub>i</sub> of 0.42 J/L using a continuous-flow system with 10 cm H₂O CPAP (average WOB<sub>i</sub> + lung work = 0.82 J/L, assumed average lung work 0.40 J/L). Using the Katz lung model, Akashi et al<sup>19</sup> noted increased WOB<sub>i</sub> with a continuous-flow system as CPAP was increased: 0.412 J/L at 5 cm H₂O CPAP and 0.705 J/L at 10 cm H₂O CPAP. In two series of patients being weaned from ventilatory support, Bey-
Table 3. Results of Bench Evaluation of Seven Ventilators at 10 cm H2O PSV and 0 cm H2O CPAP*  

<table>
<thead>
<tr>
<th></th>
<th>WOBi</th>
<th>P_{\text{max-avg}}</th>
<th>V_{\text{max}}</th>
<th>t_i</th>
<th>V_t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>J/L</td>
<td>cm H\textsubscript{2}O</td>
<td>L/min</td>
<td>s</td>
<td>mL</td>
</tr>
<tr>
<td>6400ST</td>
<td>0.0002 (0.002)\dagger</td>
<td>-0.5 (0.43)</td>
<td>47 (1)</td>
<td>0.9 (0.1)</td>
<td>542 (5)</td>
</tr>
<tr>
<td>IRISA (FR)\ddagger</td>
<td>0.001 (0.001)</td>
<td>-0.5 (0.2)</td>
<td>71 (1)</td>
<td>0.6 (0.0)</td>
<td>490 (4)</td>
</tr>
<tr>
<td>Vcolar</td>
<td>0.002 (0.000)</td>
<td>-1.3 (0.2)</td>
<td>55 (1)</td>
<td>1.0 (0.0)</td>
<td>541 (2)</td>
</tr>
<tr>
<td>7200a</td>
<td>0.006 (0.001)</td>
<td>-1.9 (0.5)</td>
<td>52 (1)</td>
<td>0.9 (0.0)</td>
<td>505 (5)</td>
</tr>
<tr>
<td>IRISA (SR)\S</td>
<td>0.008 (0.003)</td>
<td>-0.7 (0.3)</td>
<td>53 (1)</td>
<td>0.9 (0.0)</td>
<td>500 (3)</td>
</tr>
<tr>
<td>900C</td>
<td>0.010 (0.000)</td>
<td>-2.3 (0.1)</td>
<td>87 (0)</td>
<td>0.7 (0.0)</td>
<td>506 (3)</td>
</tr>
<tr>
<td>Bear 3</td>
<td>0.013 (0.001)</td>
<td>-2.1 (0.2)</td>
<td>62 (0)</td>
<td>0.8 (0.0)</td>
<td>530 (1)</td>
</tr>
<tr>
<td>Bear 5</td>
<td>0.017 (0.006)</td>
<td>-1.9 (0.2)</td>
<td>61 (1)</td>
<td>1.0 (0.0)</td>
<td>477 (3)</td>
</tr>
</tbody>
</table>

* Lung model peak flow set at 40 L/min, tidal volume at 400 mL.
\dagger All values are mean (SD).
\ddagger FR = fast response.
\S SR = slow response.

Table 4. Results of Bench Evaluation of Seven Ventilators at 10 cm H2O PSV and 10 cm H2O CPAP*  

<table>
<thead>
<tr>
<th></th>
<th>WOBi</th>
<th>P_{\text{max-avg}}</th>
<th>V_{\text{max}}</th>
<th>t_i</th>
<th>V_t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>J/L</td>
<td>cm H\textsubscript{2}O</td>
<td>L/min</td>
<td>s</td>
<td>mL</td>
</tr>
<tr>
<td>7200a</td>
<td>0.001 (0.000)\dagger</td>
<td>-0.4 (0.1)</td>
<td>45 (1)</td>
<td>0.9 (0.0)</td>
<td>540 (4)</td>
</tr>
<tr>
<td>Bear 5</td>
<td>0.012 (0.002)</td>
<td>-1.5 (0.2)</td>
<td>65 (1)</td>
<td>0.9 (0.0)</td>
<td>527 (2)</td>
</tr>
<tr>
<td>Vcolar</td>
<td>0.014 (0.005)</td>
<td>-2.0 (0.6)</td>
<td>57 (1)</td>
<td>1.1 (0.0)</td>
<td>599 (3)</td>
</tr>
<tr>
<td>900C</td>
<td>0.026 (0.002)</td>
<td>-3.8 (0.2)</td>
<td>87 (0)</td>
<td>0.8 (0.0)</td>
<td>580 (4)</td>
</tr>
<tr>
<td>6400ST</td>
<td>0.037 (0.005)</td>
<td>-2.6 (0.5)</td>
<td>60 (1)</td>
<td>0.9 (0.0)</td>
<td>554 (3)</td>
</tr>
<tr>
<td>IRISA (FR)\ddagger</td>
<td>0.038 (0.011)</td>
<td>-1.2 (0.23)</td>
<td>58 (1)</td>
<td>0.9 (0.0)</td>
<td>520 (2)</td>
</tr>
<tr>
<td>IRISA (SR)\S</td>
<td>0.059 (0.018)</td>
<td>-1.6 (0.37)</td>
<td>51 (1)</td>
<td>1.0 (0.0)</td>
<td>530 (3)</td>
</tr>
<tr>
<td>Bear 3</td>
<td>0.075 (0.004)</td>
<td>-3.6 (0.2)</td>
<td>66 (2)</td>
<td>0.8 (0.0)</td>
<td>555 (2)</td>
</tr>
</tbody>
</table>

* Lung model peak flow set at 40 L/min, tidal volume at 400 mL.
\dagger All values are mean (SD).
\ddagger FR = fast response.
\S SR = slow response.
WOB DURING CPAP AND PSV

Fig. 3. Representative pressure-volume loops from all ventilators tested when set at 0 cm H2O PSV and 0 cm H2O CPAP: (A) PPG Industries IFISA; (B) Hamilton Veolar; (C) Continuous-flow system (free standing); (D) Puritan-Bennett 7200a (Flow-by 20 L/min); (E) Intermed Bear 3; (F) Puritan-Bennett 7200a; (G) Intermed Bear 5 (continuous flow 20 L/min); (H) Newport E100i; (I) Bird 6400ST; (J) Siemens Servo 900C; (K) Intermed Bear 5; (L) Newport Breeze; (M) Puritan-Bennett 7200a (with tower).

don et al measured WOB of 0.220 J/L (Servo 900C) and 0.170 J/L (continuous flow) at 0 cm H2O CPAP, and Viale et al measured WOB of 0.277 J/L (Servo 900C) and 0.190 J/L (continuous flow) at 6.5 cm H2O CPAP.

A number of factors contribute to the differences in WOB noted among studies: (1) The use of lung models vs volunteers vs critically ill patients—with volunteers and patients, ventilatory drive and pattern may vary considerably; whereas, with lung models, the ventilatory pattern is constant. (2) Sensitivity settings varied considerably—we set sensitivity at -1 cm H2O (as did Capps et al); Fiaistro et al set it at -0.5 cm H2O, Katz et al and Beydon et al set 'maximum sensitivity;' and Viale et al and Bersten et al did not indicate what sensitivity was set. (3) The type of humidifier used—we used a passover humidifier (as did Katz et al) except where indicated. Beydon et al did not use a humidifier; Fiaistro et al and Viale et al used bubble-through humidifiers, and Capps et al did not specify whether a humidifier was used. (4) Function of the particular unit evaluated—in all of these studies, except that of Fiaistro et al in which three 7200a units were evaluated, only one ventilator of each type was evaluated. Thus, the actual function of the units evaluated may have been suboptimal. Actual function of a ventilator may vary considerably from the manufacturer's standard, even when manufacturer servicing has recently been completed or when a unit loaned by the manufacturer is used. Variation may even be noted when ventilators are evaluated directly from the assembly line. This variance can be assessed through evaluation of a large number of ventilators of each type; however, the time and cost required for such an evaluation make it difficult to perform. (5) Subtle changes in the demand-system function of specific ventilator models may be introduced by manufacturers, resulting in slightly different systems being evaluated by different research groups. We believe this may be the case with the Veolar.
and may explain why marked differences exist between our data for the Veolar and that of Capps et al.  

The differences we noted in WOB, between 0 and 10 cm H2O CPAP (0 cm H2O PSV) can be attributed to the function of exhalation valves and PEEP devices. These PEEP-producing apparatus have been classified as threshold resistors or orifice resistors. Threshold resistors generate a pressure (P) by exerting a force (F) over a discrete surface area (SA):

\[ P = F/SA. \]

Theoretically, threshold resistors generate pressure without causing flow resistance. In contrast, orifice
resistors impose resistance (R) to expiratory flow (V) by means of an adjustable orifice to establish an end-expiratory pressure (P):

\[ P = RV \]

In reality, most apparatus classified as threshold resistors actually function as partial flow resistors. Their function is better described by the equation:

\[ P = F/SA + R(\dot{V}_{exh} + \dot{V}_{sys}) \]

in which \( \dot{V}_{exh} \) is the patient's exhaled flow rate and \( \dot{V}_{sys} \) is the rate of gas flow through the system.\(^{21}\)

that is, the PEEP level is established both by the design of the valve and the gas flow through it. The greater the dependence on flow to establish PEEP in any exhalation-PEEP valve, the greater the work imposed by the valve. As inspiration occurs with an orifice resistor, the diversion of system flow from the exhalation valve to the patient lowers system end-expiratory pressure. This requires the patient to generate a greater pressure to maintain diversion of flow and results in greater WOB\(_i\). The orifice-resistor properties of threshold resistors functioning as exhalation-PEEP valves have been demonstrated by many.\(^{22-26}\)

Minimal WOB\(_i\) was noted with our freestanding CPAP system for two reasons: (1) A Vital Signs CPAP valve was used to establish CPAP. As noted by Marini et al.\(^{22}\) and Banner et al.\(^{24}\), this device offers limited flow resistance. As a result, the device functions predominantly as a threshold resistor. However, when we evaluated the MaxiPEEP valve by Boehringer (10 cm H\(_2\)O setting), using our continuous-flow system at 60 L/min continuous-flow and lung-model settings of \( V_T \) 400 mL and \( V_{max} \) 40 L/min, we noted a marked increase in WOB\(_i\) (0.596 ± 0.002 J/L) compared to the Vital Signs CPAP valve. The MaxiPEEP valve produces increased flow resistance\(^{22}\) and increased WOB\(_i\).\(^{24}\)

We ensured that the inspiratory-flow demand of the lung model was always met by setting the CPAP system's continuous flow at least 20 L/min higher than the peak lung-model flow rate.

The WOB\(_i\) of the Newport E100i and Breeze exceeded that of most systems evaluated for two reasons: (1) the limited flow capabilities of the manufacturer-designed continuous-flow systems—these systems include a limited reservoir (1.0 L) and only allow maximum continuous flows of 48 L/min (E100i) and 53 L/min (Breeze)—and (2) the orifice-resistor properties of the Newport exhalation valve. Because maximum continuous flow is limited, a marked increase in WOB\(_i\) occurs when peak inspiratory flows exceed the maximum available continuous flows.

The differences in WOB\(_i\) that we observed between 0 and 10 cm H\(_2\)O CPAP for ventilator demand systems is primarily a result of the effect of CPAP on exhalation-valve function. As with freestanding PEEP valves, all ventilator exhalation-PEEP valves possess varying levels of orifice resistance;\(^{23,25,26}\) and therefore cause an increase in WOB\(_i\) when CPAP is applied. Additional reasons for the increased WOB\(_i\) observed include:
Table 5. Comparison of Work of Breathing Induced by Six Ventilators and One Continuous-Flow CPAP System, Determined by Three Research Groups*‡, † Using the Katz et al* Lung Model

<table>
<thead>
<tr>
<th></th>
<th>Katz et al</th>
<th>Capps et al</th>
<th>Bersten et al</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>900C</td>
<td>0.045§</td>
<td>0.010</td>
<td>0.025</td>
</tr>
<tr>
<td>Veolar</td>
<td>—</td>
<td>0.245</td>
<td>0.162</td>
</tr>
<tr>
<td>7200a</td>
<td>0.105</td>
<td>0.172</td>
<td>0.108</td>
</tr>
<tr>
<td>Bear 5 (CF)‡</td>
<td>—</td>
<td>0.113</td>
<td>0.127</td>
</tr>
<tr>
<td>Bear 5 (DF†)</td>
<td>—</td>
<td>0.196</td>
<td>0.049</td>
</tr>
<tr>
<td>MA-1 (CF)</td>
<td>—</td>
<td>0.186</td>
<td>0.152</td>
</tr>
<tr>
<td>CPAP-S** (CF)</td>
<td>0.175</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

* Set sensitivity at maximum and used passover humidification.
† Set sensitivity at -1 cm H₂O but did not describe the type of humidification used (WOB values calculated from percentages presented in Reference 7).
‡ Did not describe the sensitivity that was set or the type of humidification used (WOB values determined from graphs presented in Reference 17).
§ All values for WOB, are mean and in J/L.
¶ CF = continuous flow.
‖ DF = demand flow.
** CPAP-S is a free-standing, continuous-flow CPAP system.

(1) the magnitude of the pressure spike required to activate each system—because most ventilators sense pressure internally on the inspiratory or expiratory side, large differences can be noted between pressures measured at the airway and those displayed by the machine. (In spite of sensitivity being set at -1 cm H₂O, Pmax of -2 to -3 cm H₂O was common with or without 10 cm H₂O CPAP.)

(2) the demand flow capabilities of each system—at lung-model peak flow of 40 L/min, demand flow varied between 40 L/min (7200a Flow-by, 0 cm H₂O CPAP; and IRISA, 10 cm H₂O CPAP) and 63 L/min (Servo 900C 0 cm H₂O PSV, 10 cm H₂O CPAP).

(3) the amount of work performed by the system during 0 cm H₂O PSV. As noted in Tables 1 and 2, all of the demand systems exert a low level of PSV (1.5 to 5.0 cm H₂O) even when PSV is set at 0 cm H₂O on the ventilator. This small amount of PSV decreases the overall quantity of work imposed by the system, and augments the tidal volume delivered by rapidly increasing system pressure from subbaseline to suprabaseline.

Our results differ from those of Capps et al and Bersten et al (Table 5). These authors noted increased WOB at 10 cm H₂O CPAP, compared to 0 cm H₂O CPAP, in some ventilators but not in other ventilators. We have no explanation for these differences in results. However, it is important to distinguish between the WOB of these systems at 0 and 10 cm H₂O CPAP and the total WOB (lung plus imposed) at 0 and 10 cm H₂O CPAP in select
patient groups. We observed that WOB increased as CPAP was increased from 0 to 10 cm H2O. However, a number of authors have reported a decrease in total WOB when CPAP or PEEP is applied. This decrease in total WOB is seen in patients with acute lung injury when collapsed lung is recruited, pulmonary compliance improves, and FRC returns toward normal. CPAP also decreases total WOB and oxygen consumption in COPD patients being weaned from mechanical ventilation by equilibrating alveolar and central airway pressures in the presence of auto-PEEP. Thus, our data should not be interpreted as a reason not to employ CPAP but rather as an indication that further improvement of ventilator function in the CPAP mode is needed for certain ventilators.

The Flow-by of the 7200a outperformed our free-standing continuous-flow CPAP system (10 cm H2O CPAP only) and the continuous-flow systems of the Bear 5, Newport E100i, and Newport Breeze. None of the continuous-flow systems provides demand flow; whereas, with Flow-by, demand flow is flow-triggered and the demand system is programmed to maintain system expiratory pressure, not the end-expiratory pressure minus the set sensitivity pressure. Under the same set of conditions (ie, same level of CPAP) Flow-by outperformed the normal pressure triggering of demand flow in the 7200a. Our data concerning Flow-by are consistent with those previously reported from studies of lung models, volunteers, and actual patients.

Demand flow on the Bear 5 outperformed 20 L/min continuous flow on the Bear 5 at 10 cm H2O CPAP, but not at 0 cm H2O CPAP. We believe these differences are primarily the result of the flow-resistant properties of the balloon-type exhalation valve used on the Bear 5 and the limited flow that is available when continuous flow is used.

A passover humidifier was used in all the trials of our study, except in that of the 7200a, in which both bubble-through and passover humidification (Cascade II with and without tower) were evaluated. The tower in the humidifier impeded gas flow to the lung model, which increased both the WOB and the pressure gradient required to trigger demand flow. This effect of the tower may be more pronounced in ventilator systems in which inspiration is triggered on the inspiratory side of the circuit, as opposed to systems such as the 7200a in which inspiration is triggered in the expiratory limb.

The application of 10 cm H2O PSV markedly reduced the WOB of all ventilators at both 0 and 10 cm H2O CPAP. With some ventilators, the application of 10 cm H2O CPAP to the 10 cm H2O PSV increased the WOB and increased the pressure gradient required to trigger the pressure-supported breaths. However, we believe these increases are clinically unimportant. We speculate that the addition of 10 cm H2O CPAP to the PSV alters expiratory flow resistance, and thus increases Pmax-neg and WOB. During PSV 10 cm H2O (with 0 cm H2O CPAP), all of the ventilators, except the IRISA and Bear 5, delivered volumes at least 100 ml greater than the VT set on the lung model. It is likely that the IRISA was one of the exceptions because of its limited peak-flow capabilities (45 L/min) and the fact that exhalation is triggered when peak flow is decreased to 25%.

The highest peak flows (87 L/min) were obtained with the Servo 900C (10 cm H2O PSV, 10 cm H2O CPAP), as were the greatest pressure gradients (Pmax-neg -3.8 cm H2O) required to trigger the PSV breath. Other ventilators (6400ST, Bear 3) also required a greater than -2.5 cm H2O pressure gradient to trigger inspiration, in spite of their sensitivity being set at -1 cm H2O. Although the overall function of these ventilators was basically equivalent to the others evaluated, a concern regarding the ability of patients to cycle the ventilator, especially in the presence of auto-PEEP, must be raised. In addition, as suggested by Street and Hopkinson, the greater the pressure gradient required to trigger inspiration, the greater the discomfort perceived by the patient.

Differences during PSV, with and without CPAP, on the slow-response and fast-response settings were noted with the IRISA ventilator. The WOB and Pmax-neg were least with the fast-response setting. The response time control alters the time for system peak flow to be achieved. With the slow-response setting, peak flow is achieved toward the middle of inspiration; whereas, with the fast-response setting, peak flow is achieved near the onset of inspiration. MacIntyre et al demonstrated in
a series of patients that alterations in initial peak flow affect patient WOB and tolerance of PSV. The greater the patient ventilatory drive, the greater the initial peak inspiratory flow required. In patients with weak ventilatory drive, a slower establishment of peak PSV flow is likely to be best tolerated (ie, most comfortable).35

The limitations of our study must be recognized. It was a bench study under ideal conditions. The actual clinical performance of any ventilator is dependent upon many factors, not the least of which is its maintenance. In our study, only one ventilator of each type was evaluated. Thus, variation between units of each model were not considered. Also, the function of the lung model at 10 cm H2O CPAP must be considered. With the addition of CPAP, the end-expiratory position is maintained by a compressible column of air that is prone to oscillations. Although we attempted to ensure that oscillations were eliminated during each study period, they may have been present and may have affected our results. Care must be taken when attempts are made to extrapolate these data to the clinical setting. Ideally, the performance of each ventilator used should be evaluated to ensure that it is at a level consistent with the manufacturer's recommendations and the results of this and other studies.

Conclusions

Variability exists among mechanical ventilators at any set PSV/CPAP level and for any given ventilator across varying PSV and CPAP levels. However, on the basis of our findings we can only speculate on the clinical importance of these differences. Overall, we believe that all of the ventilators we evaluated function appropriately under most clinical situations, each demonstrating both enhanced and diminished performance at select settings.

Our specific findings include: (1) The WOB1 and the maximum negative pressure gradient necessary to establish flow during spontaneous ventilation increase with the application of CPAP during either continuous or demand flow. (2) All demand systems evaluated produced low level PSV (1.5-5 cm H2O) at both 0 and 10 cm H2O CPAP, even if the PSV controls were set at 0 cm H2O. (3) The Flow-by of the 7200a outperformed the standard demand flow of the 7200a. (4) The continuous-flow option of the Bear 5 outperformed the standard demand flow of the Bear 5 at 0 cm H2O CPAP, but not at 10 cm H2O CPAP. (5) The addition of the tower to the Cascade II humidifier during use of the 7200a resulted in increased WOB; and greater pressure gradient required to trigger the demand flow than when the Cascade II was used without the tower. (6) The free-standing CPAP system performed well when compared to the demand systems at both 0 and 10 cm H2O CPAP; however, results may vary considerably with the use of specific PEEP valves. (7) The WOB1 at 0 and 10 cm H2O CPAP was reduced by the application of 10 cm H2O PSV. (8) The addition of 10 cm H2O CPAP to 10 cm H2O PSV increased the pressure gradient required to trigger the PSV on most ventilators evaluated. (9) The fast-response setting on the IRISA during PSV outperformed the slow-response setting.

PRODUCT SOURCES

Mechanical ventilators:
Intermed Bear 3 and Bear 5, Bear Medical Systems Inc,
Riverside CA
6400ST, Bird Products Corp, Palm Springs CA
7200a, Puritan-Bennett Corp, Carlsbad CA
IRISA, Biomedical Systems (currently SARA Medical Systems), PPG Industries Inc, Lenexa KS
Volar, Hamilton Medical, Reno NV
Servo 900C, Siemens Life Support Systems,
Schaumburg IL
Breeze and E100i, Newport Medical Instruments Inc,
Newport Beach CA

Monitoring and data processing equipment:
3700 pneumotachograph, Hans Rudolph Inc,
Kansas City MO
MP-45-32-871 ± 100 cm H2O differential pressure
transducer, Validyne Engineering Corp, Northridge CA
DEC LSI 11/23 computer, Digital Equipment Co,
Boston MA
ADAC data-acquisition module, ADAC Corp, Woburn MA
40006-1 graphics terminal, Tektronix, Beaverton OR

Valves:
MaxiPEEP valve, Boehringer Laboratories Inc,
Norristown PA
CPAP valve, Vital Signs Inc, Totowa NJ

Humidifiers:
Cascade I and II humidifiers, Puritan-Bennett Corp,
Carlsbad CA
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Role Conflict and Role Ambiguity among Respiratory Care Managers

George C Burke III DrPH, Lynda S Tompkins MS RRT, and John D Davis MS RRT

BACKGROUND: The health occupations and management literature does not specifically address role stress among technical directors of respiratory care departments. We undertook an analysis of role conflict, role ambiguity, and job satisfaction among technical directors of respiratory care departments in Texas. METHODS & MATERIALS: We distributed a questionnaire designed to measure role conflict and ambiguity and a questionnaire to elicit demographic and organization data to 283 technical directors in all Texas hospitals with more than 75 beds. Organization characteristics and demographic factors were studied as moderators. RESULTS: Analysis of the 199 responses received revealed that both role conflict (mean [SD] 3.86 [0.97] on 7.0 scale) and role ambiguity (2.64 [0.93] on 7.0 scale) scores were low compared to the neutral point of measure. Role overload, a component of role conflict, was found to be above the neutral point (4.64 [1.85]). One-way analysis of variance revealed no significant differences between the role conflict or the role ambiguity measures based on age, race, gender, number of employees supervised, size of institution, and position to which the respondent reported. Role conflict and role overload were each found to have significant negative correlations with job satisfaction (p < 0.01). CONCLUSION: We are encouraged by the low role-conflict and ambiguity scores observed but concerned about the elevated role-overload scores. We believe that an in-depth study of role overload among respiratory care managers is warranted. (Respir Care 1991;36:829-836.)

Background

The literature suggests that there should be a great deal of role stress among managers in hospitals generally and respiratory care specifically. Peter Drucker once claimed that hospitals are among the most complex organizations to manage.1 White and Wisdom2 echoed this opinion by stating, "The management of interpersonal relations, the importance of the services offered, and ever-changing technology in the hospital all contribute to a highly stressful environment." Rawlins reported that job stress was a concern in a study he conducted of respiratory therapists, with increased job stress related to turnover in his sample.3 Respiratory care managers must sometimes make decisions that involve a trade-off between cost and quality within an extremely complex environment. In these circumstances, role stress would be predicted. Yet a void exists in the management and health occupations literature concerning role stress among directors of respiratory care departments. The present study was conducted to begin to fill that gap.

In an organization context, role is defined as a set of expectations applied to the incumbent of a particular position by others, both within and beyond an organization's boundaries.4-6 Roles usually serve a useful purpose by prescribing for the incumbent the expectations for the job. Yet, roles can sometimes be problematic. Role stress is a general concept encompassing those conditions in which role demands are vague, difficult, or impossible to meet.7 Within the concept of role stress are two subconstructs: role conflict and role ambiguity. Role conflict has been described by Rizzo, House, and Lirtzman8 as (1) conflict between an individual's internal standards or values and the defined role behavior; (2) conflict between the time, resources,
or capabilities of the individual and the defined role behavior; (3) conflict in the form of incompatible organization policies and requests; and finally (4) role overload, meaning conflict between several roles for the same person that require different or incompatible behaviors. Burke and Scalzi state:7

In more familiar terms, role conflict is that feeling people get in the pit of their stomachs when the boss asks for the impossible, and needs it immediately, or when they are caught in a crossfire between two senior managers, or when three deadlines are staring them in the face and the family vacation starts tomorrow.

Role overload, a specific form of role conflict, appears to be present among various health care professionals. Kahn et al describe role overload:9

A very prevalent form of conflict in industrial organizations is role overload. Overload could be regarded as a kind of inter-sender conflict in which various role senders may hold quite legitimate expectations that a person perform a wide variety of tasks, all of which are mutually compatible in the abstract, but it may be virtually impossible for the local person to complete all of them within the time limits. He is likely to experience overload as a conflict of priorities.

Role ambiguity can be defined as the extent to which clarity is lacking regarding job performance expectation, methods for carrying out the job, and consequences of performance.10 A person who is given insufficient information regarding his or her organization is likely to use coping behavior and defense mechanisms to solve problems or avoid stress. Burke and Scalzi state it in these terms:7

Role ambiguity is a feeling that people get when they play a game without knowing the rules—they have to get zapped numerous times before they can play, and they know the rules can change without warning.

A number of studies have been conducted regarding role conflict and role ambiguity, with several studies specific to health care managers. We found no studies of role conflict and role ambiguity among respiratory care managers. The best documented outcomes of role conflict are job dissatisfaction and job-related tension, which have been isolated in a variety of occupational groups.8,11-16 Other studies have reported correlations between role conflict and organizationally dysfunctional outcomes such as unsatisfactory work group relationships,17 slower and less accurate group performance,18 lower commitment to the organization,19 lower performance evaluations,20 inadequate perceived leader behavior,21 less confidence in the organization,9 and unfavorable attitudes towards role senders.15 In a study of nurse executives, Scalzi21 reported that role conflict was related to increased depression and decreased job satisfaction among nursing administrators. She also reported that role overload, a specific form of role conflict, was a particular concern. In a study of directors of medical records, Floreani and Burke22 likewise found that role conflict was related to job dissatisfaction and noted the existence of role overload as a major contributor to role conflict. In a study of hospital executives, one of us (GCB)23 has reported that role conflict is associated with job dissatisfaction, that the dissatisfaction varies with the level in the organization, and that role overload is also a major contributor to role conflict.

In studies of role ambiguity, Johnson and Graen24 have linked role ambiguity to job turnover. Caplan and Jones25 have reported that lack of clarity about performance is associated with a greater concern about performance, lower actual and perceived group productivity, less involvement with the group, lower job satisfaction, and other deleterious effects.25 Brief and Aldag22 and Greene26 have reported that role incumbents with high levels of role ambiguity respond with anxiety; depression; physical symptoms; a sense of futility or lower self-esteem; lower levels of job involvement and organization commitment; and perceptions of lower performance on the part of the organization, supervisors, and themselves.

Role studies of respiratory care managers are scarce. Prewitt27 studied the role of respiratory care practitioners, with emphasis on patient management and evaluation. Rawlins3 studied job stress and satisfaction among respiratory therapists, reporting that job dissatisfaction (contributing to the decision to leave the job) increases as the magnitude of stressors in the work environment increases. In a study of respiratory care practitioners in 18 New York Hospitals, Hmelo and Axton28 concluded that the more congruent one’s job is with one’s role expectation, the more satisfied one is likely to be with the work in general. Given a void in the literature specific to role stress among directors of
respiratory care, we believe that the results of our study are important.

This study examined the perceived levels of role conflict and role ambiguity among directors of respiratory care departments and whether these perceptions varied based on organization and demographic characteristics. Specifically, we asked three research questions: (1) What are the perceived levels of role conflict (including role overload) and role ambiguity? (2) Are the perceived levels of role conflict and role ambiguity moderated by the age, race, gender, number of employees supervised, size of institution, and position to which the respondent reports? (3) What are the correlations between role conflict, role ambiguity, and job satisfaction?

Methods and Materials

Subjects

We mailed surveys to the technical directors of the 283 Texas general and acute care hospitals listed in the Texas Hospital Association Directory as having more than 75 beds. Hospitals with fewer than 75 beds were excluded because hospitals of this size often do not report statistics to the American Hospital Association's Guidebook, from which the demographic data were gathered.

Materials

The instrument used to collect data for this study was taken, in part, from an instrument developed by Rizzo et al. Part A of the questionnaire was designed to measure role conflict and role ambiguity and according to the review by Van Sell et al has been used extensively in earlier studies. Rizzo et al’s instrument is based upon Kahn’s theories and concepts of role conflict and role ambiguity. The strong theoretical basis of Kahn’s works lends credence to the content validity of Rizzo et al’s instrument.

A 7-point Likert scale was employed to measure a subject’s response to each of 14 items, with 1 representing a response of Always and 7 indicating a response of Never. The items in Part A included 8 questions that measure perceived levels of role conflict and 6 items that measure role ambiguity. Rizzo et al reported reliabilities for the 8 role-conflict items to be 0.82 for 2 samples studied.

Reliabilities for the 6 role-ambiguity items were reported to be 0.78 and 0.81.

Part B of the questionnaire consisted of demographic and organization data, age, gender, race, bed size of institution, annual budget, position to which the respondent reports, number of employees the respondent supervises, and clinical and administrative background of the supervisor.

Procedures

The instrument was mailed directly to the technical directors of the respiratory care departments of the 283 hospitals. A self-addressed envelope coded to maintain confidentiality and yet allow follow-up was included to encourage return. A second mailing was conducted in which the questionnaires were again mailed to those directors who had not responded. A t test was used to determine whether the responses differed significantly between the group who responded to the initial mailing and those responding to the second mailing.

After we received the completed questionnaires from the second mailing, all remaining non-respondents were telephoned. Responses were collected by a graduate student who coded the responses to maintain confidentiality. A t test was used to determine whether a significant difference existed in the conflict and ambiguity scores based on whether the respondent answered via mail or telephone interview. Chi-square analysis was used to determine whether a significant difference existed in the nonparametric variables based on method of answering the survey (eg, race or sex).

To facilitate analysis, the directors were grouped according to the number of employees they supervised, with 1 representing a staff with less than 12 employees, 2 representing 13 to 48 employees, and 3 representing more than 50 employees. Subjects were also grouped according to the bed size of the hospital in which they were employed, with 1 indicating 75 to 150 beds, 2 indicating 151 to 300 beds, and 3 indicating 301 to 1,500 beds. Three age groups were used for analysis. Group 1 was comprised of those subjects of the ages 26 thru 30 years of age, Group 2 those 31 thru 50 years, and Group 3 those subjects who were ≥ 51 years.
Data were analyzed using the Statistical Package for the Social Sciences (SPSS-X, SPSS Inc., Chicago IL). The Role-Conflict Score was derived for each subject by finding the mean score of the 8 items that measured role conflict. The items were 2, 4, 6, 7, 9, 11, 12, and 14. Item 4, "I work with two or more groups who operate much differently," was used to indicate role overload. One measure, the Overall Mean Conflict Score, was established as the measure of central tendency to represent all subjects’ Role-Conflict Scores. This measure was found by calculating the arithmetic average of the subjects’ Role-Conflict Scores.

The Role-Ambiguity Score for each subject was derived from the average score on the 6 Role-Ambiguity items, which were Items 1, 3, 5, 8, 10, and 13. One measure, the Overall Ambiguity Score, was established to represent all subjects’ Role-Ambiguity Scores. This measure is found by calculating the arithmetic average of the subjects’ Role-Ambiguity Scores.

Job Satisfaction was measured by response to a single item, "I am satisfied with my position as director." Directors were asked to indicate their response to this item on a 7-point Likert Scale, with 1 = True and 7 = False. The researchers recognize that a 1-item response may not, in itself, be a valid indicator of satisfaction, however, the item was included as a point of interest. Any conclusions that may include the responses to this item must consider this limitation.

Results

Of the 283 surveys mailed, 175 were returned. Because no significant difference exists between the responses of the first group and the responses of the second group, t(173) = 0.04, p = 0.966, all responses were analyzed together. The mailout response rate was 61.8%. Of the 108 attempted follow-up telephone calls, 30 were completed. Six of the 30 responses were omitted because the hospital bed size was less than 75. The 24 responses collected via telephone were not significantly different at the 0.01 a level from the responses collected by mail as shown by t-test analysis of the role conflict measure, where t(197) = 0.06, p = 0.953 and chi-square analysis of the demographic measures, with race X^2(3) = 3.30, sex X^2(1) = 0.10, age group X^2(2) = 1.68, and position to whom respondent reports X^2(5) = 8.89. Because the telephone respondents were not found to be different from the mail respondents, all responses were included in the final analysis. One hundred and thirty respondents were men (65.3%). Respondents ranged from 26 years to "over 56," with the modal age-interval being 26-30 years (84.3%). The largest proportion of the subjects were Anglo (77.9%), followed by Hispanic (18.1%), Black (3.5%), and Other (not-specified) (1.5%). The majority of the respiratory directors who responded report to an associate or assistant administrator (54.8%).

The directors indicated that they supervise staffs of sizes ranging from 1 employee to 200 employees, with the modal staff size 15 employees. The size of the hospitals (based on number of beds) represented by these directors ranges from 75-1500 beds, with the modal bed size 150.

The Role-Conflict Scores ranged from 1.5 to 6.38 (with the low score, 1.0, representing minimum role

Table 1. Summary of Responses of 199 Respiratory Care Technical Directors to Role-Conflict Items

<table>
<thead>
<tr>
<th>Questionnaire Item</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. I receive assignments without the manpower to complete them.</td>
<td>3.74</td>
<td>1.58</td>
</tr>
<tr>
<td>4. I work with two or more groups who operate quite differently.</td>
<td>4.64</td>
<td>1.85</td>
</tr>
<tr>
<td>6. I have to buck a rule in order to carry out an assignment.</td>
<td>3.37</td>
<td>1.69</td>
</tr>
<tr>
<td>7. I receive incompatible requests from two or more people.</td>
<td>3.72</td>
<td>1.49</td>
</tr>
<tr>
<td>9. I do things that are apt to be accepted by one person and not accepted by another</td>
<td>4.35</td>
<td>1.65</td>
</tr>
<tr>
<td>11. I have to do things that should be done differently.</td>
<td>3.89</td>
<td>1.66</td>
</tr>
<tr>
<td>12. I work on unnecessary things.</td>
<td>3.56</td>
<td>1.61</td>
</tr>
<tr>
<td>14. I receive an assignment without adequate resources and materials to execute it.</td>
<td>3.59</td>
<td>1.57</td>
</tr>
</tbody>
</table>

Mean Role-Conflict Score 3.86 0.97
stress and the high score, 7.0, representing maximum role stress). The Overall Mean Conflict Score for the directors was 3.86 (SD = 0.97). The means and standard deviations for each item expressing role conflict and the Overall Mean Role-Conflict Score are illustrated in Table 1.

The role overload measure (Item 4) was found to be the highest score of all items, with the \( \bar{x} = 4.64, \) SD = 1.85. This item and Item 9, “I do things that are apt to be accepted by one person and not accepted by another” \( (\bar{x} = 4.35, \) SD = 1.65), were the only 2 items that scored above the neutral point of 4.0.

The Role-Ambiguity Scores ranged from 1.0 to 6.0, with the low score (1.0) indicating low role ambiguity and the high score (7.0) representing high role ambiguity. The Overall Mean Role-Ambiguity Score for the directors was 2.64 (SD = 0.93). The means and standard deviations for each role-ambiguity item and the Overall Mean-Ambiguity Score are listed in Table 2.

Table 2. Summary of Responses of 199 Respiratory Care Technical Directors to Role-Ambiguity Items

<table>
<thead>
<tr>
<th>Questionnaire Item</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I know what my responsibilities are.</td>
<td>1.77</td>
<td>1.08</td>
</tr>
<tr>
<td>3. I have clear planned goals for my job.</td>
<td>2.42</td>
<td>1.18</td>
</tr>
<tr>
<td>5. I know I have divided my time properly.</td>
<td>3.33</td>
<td>1.35</td>
</tr>
<tr>
<td>8. I feel certain about how much authority I have.</td>
<td>2.53</td>
<td>1.51</td>
</tr>
<tr>
<td>10. I know exactly what is expected of me.</td>
<td>2.44</td>
<td>1.32</td>
</tr>
<tr>
<td>13. Explanations are clear of what has to be done.</td>
<td>3.31</td>
<td>1.40</td>
</tr>
</tbody>
</table>

| Mean Role-Ambiguity Score                            | 2.64 | 0.93|

The majority of directors (84.8%) responded positively to the job satisfaction item, with scores of 1.2, or 3 \( (1 = \) True, higher satisfaction; \( 7 = \) False, lower satisfaction). Only 17 directors indicated a response greater than the neutral point of 4.0. Six directors indicated the extreme right score of 7.0. The average job satisfaction score for the subjects was 2.33 (SD = 1.45).

One-way analysis of variance revealed no significant differences between the Overall Role-Conflict Scores based on age, race, sex, number of employees supervised, size of institution, or position to which directors report, at \( \alpha = 0.01 \) (Table 3). Analysis also indicated no significant difference between the Overall Role-Ambiguity Scores based on the same groups, at \( \alpha = 0.01 \) (Table 4).

Pearson’s product moment correlation was performed using three variables: (1) Conflict Scores, (2) Ambiguity Scores, and (3) Job Satisfaction Scores. The resulting correlations are listed in Table 5. The Conflict Scores and Ambiguity Scores showed a significant positive relationship \( (r = 0.399, p < 0.01) \) with each other. The Conflict Scores and Ambiguity Scores each showed a significant negative correlation with the Job Satisfaction measure \( (r = -0.310, p < 0.01, \) and \( r = -0.429, p < 0.01, \) respectively).

Discussion

These directors of respiratory care departments demonstrated relatively low role-conflict scores (below neutral), with even lower role-ambiguity scores. The low ambiguity scores are consistent with previous research involving other health care professionals (eg, directors of medical records departments\(^{22}\) and nurse executives and hospital executives\(^{7}\)). The low conflict scores are not consistent with medical records directors, who scored above the neutral point,\(^{22}\) or with nurse executives who scored a value equal to the neutral point.\(^{7}\) This study failed to show a difference in the role-conflict scores or the role-ambiguity scores based on age, race, sex, number of employees supervised, size of institution, or position to which directors report.

The low role-ambiguity scores suggest that respiratory care directors, as a group, do not appear to struggle with the behavioral requirements of their position. Directors appear to have a clear understanding of their specified set of tasks and their responsibilities within the health care industry.

The low role-conflict scores may indicate that, on the average, respiratory care directors do not experience stress related to a conflict between their personal standards and their defined role behavior. However, an interesting finding is that although the directors exhibited rather low role-conflict scores, they also expressed high (above neutral) role-overload scores. These findings are consistent with...
Table 3. Analysis of Variance of the Overall Mean-Conflict Score of 199 Technical Directors of Respiratory Care Departments in Texas

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Between groups</td>
<td>2</td>
<td>0.096</td>
<td>0.100</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>195</td>
<td>0.959</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>197</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>Between groups</td>
<td>3</td>
<td>0.268</td>
<td>0.280</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>195</td>
<td>0.958</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>198</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>Between groups</td>
<td>1</td>
<td>0.252</td>
<td>0.265</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>197</td>
<td>0.951</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>198</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of employees</td>
<td>Between groups</td>
<td>2</td>
<td>0.581</td>
<td>0.609</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>195</td>
<td>0.953</td>
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<tr>
<td></td>
<td>Total</td>
<td>197</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital size</td>
<td>Between groups</td>
<td>2</td>
<td>0.294</td>
<td>0.306</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>194</td>
<td>0.959</td>
<td>Total</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>196</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Position to report</td>
<td>Between groups</td>
<td>5</td>
<td>1.746</td>
<td>1.88</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>193</td>
<td>0.927</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>198</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No significant difference at $\alpha = 0.01$.

previous research in which role overload was found to be high among hospital executives, nurse executives,7 and medical records directors.22

The existence of role overload may indicate that respiratory care directors (as with the other health care professionals previously studied) may not have difficulty managing the demands of their role, in general; however, the volume of demands and the stringent time frame imposed to meet those demands may potentiate role stress. Within the patient care environment, respiratory care directors find themselves in a multifaceted role that requires the individual to act as clinician (many times providing direct patient care), financial manager (providing state-of-the-art medical equipment within an acceptable budget), staff manager (developing effective schedules to provide 24-hour patient care), nurse liaison (establishing and maintaining a "team" approach to patient care), and physician liaison (developing a collegial working relationship with physicians while fulfilling orders). All of these expectations may cause the director to feel as if he or she is being "bombarded" with thousands of duties that must be accomplished on a daily basis. The identification of the overwhelming number of demands may help explain the directors' elevated role-overload score.

The responses of the subjects were summarized as means even though the instrument used a Likert scale, for which the median may be a more appropriate measure of central tendency. This decision was based on the assumption that the responses represented an underlying metric. The mean allowed the use of more powerful parametric statistics.

This study is limited by the 1-statement job satisfaction measure. Future studies should consider incorporating a multi-item instrument to better measure job satisfaction.

Organizations that operate with a clear chain of command and record specific job descriptions should be able to provide a more satisfactory work environment for employees. Although the role conflict and ambiguity scores for this group were
Table 4. Analysis of Variance for the Overall Mean-Ambiguity Score of 199 Respiratory Care Technical Directors in Texas

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
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<td>Age</td>
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<td>0.062</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>195</td>
<td>0.872</td>
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</tr>
<tr>
<td></td>
<td>Total</td>
<td>197</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>Between groups</td>
<td>3</td>
<td>1.28</td>
<td>1.50</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
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<td>0.853</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>Between groups</td>
<td>1</td>
<td>0.312</td>
<td>0.361</td>
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<tr>
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<td>Within groups</td>
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</tr>
<tr>
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<td>Total</td>
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<td></td>
</tr>
<tr>
<td>Number of employees</td>
<td>Between groups</td>
<td>2</td>
<td>0.222</td>
<td>0.255</td>
</tr>
<tr>
<td></td>
<td>Within groups</td>
<td>195</td>
<td>0.870</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
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<td></td>
</tr>
<tr>
<td>Hospital size</td>
<td>Between groups</td>
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<td>1.65</td>
<td>1.94</td>
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<tr>
<td></td>
<td>Within groups</td>
<td>194</td>
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<td></td>
<td>Total</td>
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<td></td>
</tr>
<tr>
<td>Position to report</td>
<td>Between groups</td>
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<td>1.52</td>
<td>1.80</td>
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<tr>
<td></td>
<td>Within groups</td>
<td>193</td>
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</tr>
<tr>
<td></td>
<td>Total</td>
<td>198</td>
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</tr>
</tbody>
</table>

No significant difference at $\alpha = 0.01$.

Table 5. Pearson Correlation for Conflict, Ambiguity, and Job Satisfaction Scores

<table>
<thead>
<tr>
<th>Variables</th>
<th>n</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conflict with ambiguity</td>
<td>199</td>
<td>0.399*</td>
</tr>
<tr>
<td>Conflict with satisfaction</td>
<td>197</td>
<td>-0.310*</td>
</tr>
<tr>
<td>Ambiguity with satisfaction</td>
<td>197</td>
<td>-0.429*</td>
</tr>
</tbody>
</table>

* $p < 0.01$.

low, the general tendency of scores appeared as expected: As role conflict and role ambiguity increase, job satisfaction decreases.

Conclusions

Respiratory care is a highly technical, ever-changing profession in which managers may find themselves caught up in a deluge of daily activities that must be performed ‘right away.’ The low role conflict and ambiguity scores found with this group are encouraging. However, indications pointing to elevated role overload are troublesome. A more in-depth study of role overload among health care professionals is warranted. Identification of the specific components of overload and job satisfaction may prove enlightening.

REFERENCES

23. Burke GC. Understanding the dynamic role of the hospital executive: the view is better from the top. Hosp Health Serv Adm 1988;34:99-112.
27. Prewitt MW. Role expectations of the respiratory care practitioner as reported by the role incumbent and other health care professionals. Respir Care 1984;29:900-904.
An Evaluation of the Usefulness of End-Tidal $P_{CO_2}$
To Aid Weaning from Mechanical Ventilation
following Cardiac Surgery

Dean Hess MEd RRT, Amy Schlottag MSN RN, Bradley Levin MD,
John Mathai MD, and William O Rexrode MD

We evaluated the use of end-tidal $P_{CO_2}$ ($P_{ETCO_2}$) as an indication of changes in $P_{ACO_2}$ in 24 adult patients being weaned from mechanical ventilation following cardiac surgery. METHODS: Patients were weaned by synchronous intermittent mandatory ventilation (SIMV) and T-piece trials. After each change in the weaning process (SIMV rate, T-piece trial), arterial blood gases were obtained from an indwelling catheter, and the $P_{ETCO_2}$ and respiratory rate were recorded. All patients were hemodynamically stable, and all pulse oximeter saturations were $\geq 90\%$ throughout the weaning period. A total of 113 data sets were collected. RESULTS: The correlation between $P_{ACO_2}$ and $P_{ETCO_2}$ was $r = 0.82$, although the $P_{ACO_2}$ overestimated the $P_{ETCO_2}$ by $4.0 \pm 3.7$ torr. There was no significant difference between changes in $P_{ACO_2}$ and changes in $P_{ETCO_2}$ ($p = 0.63$). However, in 43% of cases the change in $P_{ETCO_2}$ incorrectly indicated the direction of change in $P_{ACO_2}$. When $P_{CO_2}$ changes of $\geq 5$ torr occurred, $P_{ETCO_2}$ incorrectly indicated the direction of change in 30% of the cases. The respiratory rates displayed by the capnograph ($15 \pm 5$/min) were similar to the patients' actual respiratory rates ($16 \pm 5$/min). CONCLUSIONS: Although changes in $P_{ETCO_2}$ were not statistically different from changes in $P_{ACO_2}$, $P_{ETCO_2}$ did not precisely indicate changes in $P_{ACO_2}$ during weaning from mechanical ventilation following cardiac surgery. Based on these results, we do not recommend the routine use of $P_{ETCO_2}$ as a noninvasive indicator of $P_{ACO_2}$ during weaning from mechanical ventilation following cardiac surgery. (Respir Care 1991;36:837-843.)

Introduction

In recent years, there has been increasing use of noninvasive monitoring in respiratory care, with particular interest in pulse oximetry and capnography. Following cardiac surgery, patients are typically ventilated until they are hemodynamically stable and have adequately recovered from operative anesthesia. They are then weaned from the ventilator, often by use of synchronous intermittent mandatory ventilation (SIMV) and spontaneous breathing trials by T-piece. Weaning may be rapid or prolonged, and typically it involves many arterial blood gas measurements. To decrease the number of arterial blood gases required (and the related cost), we have been interested in the potential use of end-tidal $P_{CO_2}$ ($P_{ETCO_2}$) to indicate $P_{ACO_2}$ during weaning in these patients. Because the literature related to its use during ventilator weaning is sparse and conflicting, we wanted to objectively evaluate this use of capnography before incorporating it into routine use in patients following cardiac surgery. Therefore, we conducted a study to evaluate the ability of $P_{ETCO_2}$ to indicate changes in $P_{ACO_2}$ during the entire course of weaning from mechanical ventilation following cardiac surgery.

Methods

Twenty-four patients were included in the study following cardiac surgery (21 male, 3 female, age range 41-74 y). Patients were prospectively and
sequentially entered into the study, per the availability of the research team for data collection. None of the patients had a known history of pulmonary disease. For each patient, the first $P_{\text{etCO}}$ was recorded when a blood gas was obtained with the patient on full ventilatory support, at the time when the decision was made to initiate weaning. Weaning was initiated when the patients were hemodynamically stable and had acceptable conventional weaning parameters. Weaning was begun at $17.7 \pm 2.7$ hours following surgery (range 12-23 h). Subsequent to that, $P_{\text{etCO}}$ was recorded when each blood gas was obtained until the patient was extubated. Ventilator weaning was directed by the attending physician using blood gas results and clinical assessment, and was not influenced by the collection of data for this study. No arterial blood gases were obtained solely for the purposes of this study, and data collection did not affect the care otherwise received by the patients. Patients were weaned using a combination of SIMV and T-piece trials, as deemed appropriate by the attending physician. This involved decreases of 2-4 breaths/min by SIMV, followed by a trial of T-piece breathing, and then extubation. All patients were ventilated using a BEAR 1 or a BEAR 2 ventilator (Bear Medical, Riverside CA).

All blood gas samples were obtained from a radial artery catheter using standard procedure, and were obtained 30-60 min after each step in the weaning process. In brief, flush solution was cleared from the catheter and connecting tubing, after which arterial blood was anaerobically collected into a heparinized syringe. The blood was immediately placed in an ice water bath, delivered to the blood gas laboratory, and analyzed promptly. Blood gas analysis was conducted in duplicate using Ciba-Corning Model 178 analyzers (Ciba-Corning, Medfield MA). Appropriate calibration and quality control procedures for blood gas analyzers were observed. Patient temperature, mean arterial blood pressure, and heart rate were recorded at the time the blood gas was obtained, and all $P_aCO_2$ values were corrected to the patient’s body temperature.

Capnography and pulse oximetry were performed using an Ohmeda 4700 OxyCap (Ohmeda, Louisville CO). This is a sidestream capnograph with a sample rate of 150 mL/min; it also measures $F_iO_2$ and provides $O_2$ compensation. Pulse oximetry was monitored using a finger probe. The maximal $P_{etCO}$ and respiratory rate displayed on the capnograph were recorded when each blood gas was obtained. The capnogram was used to validate the $P_{etCO}$ reading. Respiratory rate by inspection and/or palpation was also recorded at this time. The capnograph was appropriately calibrated and used according to manufacturer's specifications.

A total of 113 data sets were collected.

Statistical Analysis

All data are reported as mean ± standard deviation (SD). The relationship between $P_{etCO}$ and $P_{aCO_2}$ was determined using correlation and regression analysis. Correlation analysis was performed for individual patient data, as well as the pooled data from all patients. The $P_{etCO}$-$P_{aCO_2}$ difference was calculated by subtracting $P_{etCO}$ from $P_{aCO_2}$. The difference between $P_{aCO_2}$ and $P_{etCO}$ was evaluated using Student’s paired $t$ test. For each series of blood gases obtained when weaning each patient, the change in $P_{aCO_2}$ and the change in $P_{etCO}$ were determined by subtracting the $P_{aCO_2}$ from its previous value, and the $P_{etCO}$ from its previous value. The relationship between the change in $P_{aCO_2}$ and the change in $P_{etCO}$ was determined by correlation analysis, and the difference between the change in $P_{aCO_2}$ and the change in $P_{etCO}$ was evaluated by Student’s paired $t$ test. Statistical analysis was conducted using standard methodology and commercially available software (SPSS/PC+, SPSS Inc. Chicago IL); $p \leq 0.05$ was considered significant.

Results

Individual patient data are displayed in Table 1. During weaning from the ventilator, the patients’ mean arterial blood pressure was 83 ± 9 torr (range 64-103 torr), their heart rate was 86 ± 11 beats/min (range 59-111), and their pulse oximeter saturation was 98 ± 2% (range 90%-100%). All patients were hemodynamically stable during the weaning period, and all were successfully weaned to extubation (weaning time of 2-8 h). The number
**P_{\text{etCO}_2}** IN WEANING FROM POST-CARDIAC SURGERY MV

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Surgery</th>
<th>Ventilator Hours</th>
<th>Weaning Hours</th>
<th>Number of Comparison</th>
<th>Correlation (r) for P_{\text{etCO}<em>2}/P</em>{\text{aCO}_2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>56</td>
<td>CABG</td>
<td>19</td>
<td>4.5</td>
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<td>0.88</td>
</tr>
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<td>2</td>
<td>M</td>
<td>62</td>
<td>CABG</td>
<td>19</td>
<td>6.0</td>
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<td>0.88</td>
</tr>
<tr>
<td>3</td>
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<td>5</td>
<td>0.02</td>
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<td>CABG</td>
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<td>0.98</td>
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<td>0.95</td>
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<td>CABG</td>
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<td>9</td>
<td>0.95</td>
</tr>
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<td>64</td>
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<td>0.91</td>
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<td>CABG</td>
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<td>5</td>
<td>0.32</td>
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</tbody>
</table>


Of $P_{\text{aCO}_2}$ and $P_{\text{etCO}_2}$ comparisons per patient ranged from 3 to 9.

For the 113 comparisons made of $P_{\text{aCO}_2}$ and $P_{\text{etCO}_2}$, the mean $P_{\text{aCO}_2}$ was 35 ± 6 torr (range 18-45), and the mean $P_{\text{etCO}_2}$ was 31 ± 6 torr (range 15-41) ($p < 0.001$). The mean $P_{\text{aCO}_2}$-$P_{\text{etCO}_2}$ difference was 4.0 ± 3.7 torr (range -5 torr to 16 torr). The correlation between $P_{\text{aCO}_2}$ and $P_{\text{etCO}_2}$ was $r = 0.82$; the slope of the regression line was 0.9 and the intercept was $-0.5 (P_{\text{etCO}_2} = 0.9P_{\text{aCO}_2} - 0.5)$ (Fig. 1).

We were able to evaluate 89 changes in $P_{\text{etCO}_2}$ against changes in $P_{\text{aCO}_2}$. Changes in $P_{\text{etCO}_2}$ ranged from a decrease of 9 torr to an increase of 17 torr, and changes in $P_{\text{aCO}_2}$ ranged from a decrease of 10 torr to an increase of 17 torr (Fig. 2). The correlation between changes in $P_{\text{etCO}_2}$ and $P_{\text{aCO}_2}$ was $r = 0.69$, and there was no statistically significant difference between changes in $P_{\text{etCO}_2}$ and the associated changes in $P_{\text{aCO}_2}$ ($p = 0.63$). However, in 38 of 89 cases (43%), the change in $P_{\text{etCO}_2}$ incorrectly indicated the direction of change in $P_{\text{aCO}_2}$ (Fig. 2). If a change in $P_{\text{CO}_2}$ of $\geq 5$ torr is considered clinically important, then the $P_{\text{etCO}_2}$ incorrectly indicated the direction of change in $P_{\text{aCO}_2}$ in 14 of 46 cases (30%). In 21 patients (88%), there was at least one change in $P_{\text{etCO}_2}$ that incorrectly indicated the direction of change in $P_{\text{aCO}_2}$. In 9 patients (38%), there was a change in $P_{\text{CO}_2}$ of $\geq 5$.
torr in which the $P_{etCO_2}$ failed to correctly indicate the direction of change of $P_{aCO_2}$.

The respiratory rates displayed by the capnograph (15 ± 5 breaths/min) were similar to the patients’ actual respiratory rates (16 ± 5 breaths/min). We encountered no significant problems with the use of the capnograph, and we had no difficulty obtaining valid $P_{etCO_2}$ readings.

**Discussion**

Overall, we found acceptable correlation between single-point comparisons of $P_{etCO_2}$ and $P_{aCO_2}$. Further, the $P_{aCO_2}-P_{etCO_2}$ differences in our study were similar to those reported by others. However, we were disappointed in the inability of $P_{etCO_2}$ to precisely indicate changes in $P_{aCO_2}$. The high number of cases in which $P_{etCO_2}$ incorrectly indicated the direction of change in $P_{aCO_2}$ was unacceptable. Based upon these results, we do not use $P_{etCO_2}$ to indicate changes in $P_{aCO_2}$ during weaning from mechanical ventilation following cardiac surgery. However, our results might have been different with another patient population, and more work is needed to evaluate this.

The topic of capnography has been reviewed in detail elsewhere. $P_{etCO_2}$ presumably reflects alveolar $P_{CO_2}$ ($P_{ACO_2}$). The $P_{ACO_2}$ of a single alveolus depends upon its V-Q ratio and can be as low as the inspired $P_{CO_2}$ or as high as the mixed-venous $P_{CO_2}$. $P_{etCO_2}$ is the mixture of the $P_{ACO_2}$ of those alveoli emptying at end-exhalation. The apparent inability of $P_{etCO_2}$ to precisely indicate $P_{aCO_2}$ in our
patients reflects changes in the V/Q of these patients during the weaning period. This may have been due to either changes in ventilation or changes in pulmonary blood flow. Whether due to changes in ventilation or blood flow, the resultant effect on V/Q may explain the disappointing ability of changes in $P_{\text{etCO}}$ to precisely reflect changes in $P_{\text{aco}}$. Most likely, there were numerous physiologic reasons why $P_{\text{etCO}}$ incorrectly indicated changes in $P_{\text{aco}}$ in many of our cases, and the identification of all of those reasons was beyond the scope of this paper. The relatively poor ability of $P_{\text{etCO}}$ to indicate changes in $P_{\text{aco}}$ may also have been due to inaccuracy of the capnograph or the blood gas analyzer; however, we believe that this was unlikely because all instruments were appropriately calibrated per manufacturers' instructions. In spite of this, errors inherent in the technology of capnograph may have contributed to our findings.\textsuperscript{15}

Healey et al\textsuperscript{1} studied 20 patients being weaned from mechanical ventilation. They compared the changes in $P_{\text{etCO}}$ and $P_{\text{aco}}$ after switching the patients from assist/control mode to spontaneous breathing. There was an increase in $P_{\text{aco}}$ of $\geq 10$ torr in 9 of the 20 patients, and $P_{\text{etCO}}$ rose by at least 5 torr in 7 of these patients. From the data reported by them, however, it can be seen that the change in $P_{\text{etCO}}$ incorrectly indicated the change in $P_{\text{aco}}$ in five cases (25%).

Niehoff et al\textsuperscript{2} evaluated the use of capnography during weaning in 40 patients. They found that $P_{\text{etCO}}$ had a very low sensitivity (28%) in the detection of hypercarbia ($P_{\text{aco}} > 45$ torr); in a number of their cases, the $P_{\text{aco}}$ was $> 60$ torr and the $P_{\text{etCO}}$ was $< 40$ torr. They also found that a group of post-cardiac surgery patients randomized to a capnography/oximetry monitoring group had fewer arterial blood gas determinations than a control group weaned with periodic arterial blood gas sampling.

Withington et al\textsuperscript{3} evaluated the use of capnography during weaning of 40 patients following cardiac surgery. In contradiction to our findings, they concluded that capnography provided sufficiently reliable monitoring during weaning. They reported a sensitivity of 78.6% for detection of hypercapnia using $P_{\text{aco}}$; the false-negative rate was thus 21.4% (ie, the capnograph failed to indicate hypercapnia in over 21% of the cases when it was present). They also found modest correlations between $P_{\text{etCO}}$ and $P_{\text{aco}}$ in the range of 0.67-0.81.

In a study of 20 critically ill, mechanically ventilated adult patients, Hoffman et al\textsuperscript{16} found that $P_{\text{etCO}}$ was a misleading substitute for trends in $P_{\text{aco}}$. In that study, they assessed 116 changes of $P_{\text{etCO}}$ in indicating $P_{\text{aco}}$ after changes in minute ventilation. Interestingly, 42% of the changes in $P_{\text{etCO}}$ in that study failed to correctly indicate the direction of change in $P_{\text{aco}}$ (the same proportion of incorrect changes that we found). In four of the 20 patients in that study, the trend in $P_{\text{etCO}}$ was in the opposite direction of the $P_{\text{aco}}$ (r was negative for the relationship between $P_{\text{aco}}$ and $P_{\text{etCO}}$). Raemer et al\textsuperscript{17} also found that estimation of $P_{\text{aco}}$ by monitoring $P_{\text{etCO}}$ lacked precision in patients during anesthesia. The patients in these studies, however, differed from ours because those patients were not being weaned from mechanical ventilation.

Russell et al\textsuperscript{18} evaluated the stability of the $P_{\text{aco}}$-$P_{\text{etCO}}$ difference in patients following cardiac surgery. They evaluated the relationship between $P_{\text{aco}}$ and $P_{\text{etCO}}$ from the time of admission to the intensive care unit to the time of extubation, and found wide variability of the $P_{\text{aco}}$-$P_{\text{etCO}}$ in individual patients. For many of the individual patients in that study, the relationship between $P_{\text{aco}}$ and $P_{\text{etCO}}$ did not produce a significant correlation. The relationship between $P_{\text{aco}}$ and $P_{\text{etCO}}$ was not affected by ventilator rate (which ranged from 0 to 18 breaths/min), by hemodynamics, or by pharmacologic agents administered. They concluded that the variations of $P_{\text{aco}}$-$P_{\text{etCO}}$ in individual patients do not allow capnography to be substituted for periodic arterial blood gas measurements in this patient population.

Two studies have evaluated differences between $P_{\text{etCO}}$ during ventilator breaths versus spontaneous breaths when using IMV. Weinger and Brimm\textsuperscript{9} found that $P_{\text{etCO}}$ varied widely from breath to breath during IMV. In that study, the $P_{\text{etCO}}$ of spontaneous breaths was greater than that of ventilator breaths about two thirds of the time, and they recommended that maximal $P_{\text{etCO}}$, independent of breathing pattern, be used. Smith et al\textsuperscript{10} recommended that breath-specific or maximal $P_{\text{etCO}}$ be used during weaning from mechanical ventilation. Both of these


studies, however, evaluated the single-point relationship between $P_{\text{etCO}_2}$ and $P_{acO_2}$, and neither of them evaluated the use of $P_{acO_2}$ to indicate changes in $P_{acO_2}$ during weaning. It is also of interest to note that Russell et al found that ventilator rate during weaning did not affect the $P_{acO_2}$-$P_{\text{etCO}_2}$ difference.\(^\text{18}\)

In evaluation of the individual patient data (Table 1), the correlation coefficient was less than 0.70 in 7 patients (29%). Because $r^2$ explains the variability in $P_{\text{etCO}_2}$, that can be explained by variability in $P_{acO_2}$,\(^\text{19}\) an $r < 0.70$ means that $< 50\%$ of the variability in $P_{\text{etCO}_2}$ can be explained by variability in $P_{acO_2}$. In one of our patients (#18), there was a negative correlation between $P_{\text{etCO}_2}$ and $P_{acO_2}$.

It might be argued that our data indicate that $P_{acO_2}$ correctly indicate changes in $P_{acO_2}$ in some patients. Although this is correct, unfortunately it is of limited clinical usefulness. We know of no way to predict which patients will have an acceptable relationship between $P_{\text{etCO}_2}$ and $P_{acO_2}$. Therefore, only in retrospect can one know whether the relationship between $P_{\text{etCO}_2}$ and $P_{acO_2}$ was satisfactory.

A criticism of our results might be that a number of the $P_{CO_2}$ changes were small ($< 5$ torr). However, this is the clinical reality of weaning patients from mechanical ventilation following cardiac surgery. Further, $P_{\text{etCO}_2}$ incorrectly indicated changes in $P_{acO_2}$ in 30% of the cases (38% of the patients) when $P_{CO_2}$ changes $< 5$ torr were excluded from consideration. Although it might be argued that the usefulness of $P_{\text{etCO}_2}$ is to detect large changes in $P_{acO_2}$, this is not supported by our data, those of Niehoff et al,\(^\text{2}\) or those of Hoffman et al.\(^\text{16}\) Much of the attractiveness of noninvasive monitoring such as capnography is to detect changes that are not otherwise clinically apparent: the usefulness of $P_{\text{etCO}_2}$ to detect large changes in $P_{acO_2}$ that can be readily detected by other means (e.g., respiratory rate, neurologic status, hemodynamic status) is questionable.

Use of noninvasive monitoring in respiratory care has increased dramatically in recent years, even though clear guidelines and indications for appropriate use of this technology are lacking. There has been a tendency to use this monitoring because it is technically feasible and noninvasive, rather than because it has been shown that this results in improved patient care. We found that $P_{acO_2}$ correctly indicated changes in $P_{CO_2}$ approximately 30-40% of the time and, therefore, did not aid weaning from mechanical ventilation following cardiac surgery. Until improvements occur in noninvasive technology, it may be more appropriate and cost-effective to use clinical skills and arterial blood gas measurements during weaning. Further work is needed to evaluate the appropriate role of noninvasive monitoring, clinical skills, and arterial blood gases during weaning from mechanical ventilation, particularly in patients other than postcardiac surgery patients.

ACKNOWLEDGMENTS

We thank the post-cardiac surgery nursing and respiratory therapy staff for their assistance with the data collection.

REFERENCES

P_{\text{etCO}} \text{ IN WEANING FROM POST-CARDIAC SURGERY MV}


CORRECTION

An error occurred in Hess's paper "Neonatal and Pediatric Respiratory Care: Some Implications for Adult Respiratory Care Practitioners" (Respir Care 1991;36:489-513). Part of Reference 297 was omitted; it appears below in its entirety. We regret the error.

An Evaluation of the Effectiveness of Secretion Removal with the Ballard Closed-Circuit Suction Catheter

Mary Tuleya Witmer RRT, Dean Hess MEd RRT, and Mark Simmons MS RRT RPFT

We conducted this study to compare the quantity of secretions removed with a closed-circuit suction catheter (Ballard Trach-Care) to that removed with a conventional suction catheter. METHODS: Adult patients receiving chest physiotherapy at 4- or 6-hour intervals were studied. For two consecutive treatments, they were suctioned with a Ballard catheter during one treatment and with a conventional catheter during the other treatment. The order was randomly assigned, and both catheters were used on the same shift. A suction pressure of 120 torr and size 14-Fr catheters were used. All sputum obtained during each treatment was collected in a Luken’s trap, and the mass of the secretions was determined with an O-Haus Cent-O-gram balance. Twenty-eight comparisons of results with the two catheters were made in 25 patients (16 men, 9 women; median age 59 yr, median 8 days of intubation). The median PEEP was 3 cm H2O (range 0-10); the median FiO2 was 0.45 (range 0.30-0.80). RESULTS: There was no significant difference between the quantities of secretions removed with the Ballard closed-circuit catheter (median 1.7 g) and with the conventional catheter (1.9 g) (p = 0.88). CONCLUSIONS: The results of this short-term study suggest that the Ballard catheter removes secretions as effectively as does a conventional catheter. Further work is needed to evaluate the Ballard’s effectiveness during prolonged use—and the cost of using the Ballard versus a conventional suction catheter. (Respir Care 1991;36:844-848.)

Introduction

Suctioning of secretions from the lower respiratory tract is a necessary and beneficial procedure in the care of critically ill, intubated, mechanically ventilated patients. Both off-ventilator and on-ventilator suctioning techniques can be used. With the off-ventilator technique, a catheter is passed into the airway after the patient is removed from the ventilator. With the on-ventilator technique, the catheter is passed into the airway without removing the patient from the ventilator.

Two variations of the on-ventilator technique are available. One of these involves the use of a conventional catheter passed through the swivel port of the ventilator. Although this method may prevent complications related to removal of the patient from the ventilator, it may increase contamination of the patient’s lower respiratory tract due to contaminants present on the swivel adapter. The other on-ventilator method involves the use of a closed-system suction catheter. With this method, the suction catheter system becomes part of the ventilator circuit, and the same catheter is used for a period of 24 hours.

A commonly used closed-system suction catheter is the Ballard Trach-Care (Ballard Medical Products, Midvale UT). With this system, the catheter is enclosed in a plastic sheath, which is connected to a specially designed T-piece attached to the airway. The T-piece contains a washer to prevent gas leakage around the catheter and to remove secretions from the outside of the catheter as it is withdrawn from the airway. A port is present on the T-piece to allow rinsing of the catheter and instillation of irrigating solutions. A thumb port on
the proximal end of the catheter allows suction control, and this port can be locked off to prevent accidental application of suction.

In our critical care units, we occasionally use the Ballard system on patients who poorly tolerate disconnection from the ventilator for suctioning. These patients include those on high F\textsubscript{1\textsubscript{O}}\textsubscript{2}, and PEEP settings, those with hemodynamic instability, and those with elevated intracranial pressures. A frequently expressed concern of our critical care therapists and nurses has been that the Ballard catheter is more difficult to manipulate within its sheath than a standard catheter, and thus it may be less effective in the removal of secretions. In this study, we addressed that concern by comparing the quantity of secretions removed with a Ballard catheter to the quantity removed with a conventional suction catheter.

**Methods**

The subjects in the study were 25 adult, intubated, mechanically ventilated patients in the critical care unit. There were 16 men and 9 women, with a median age of 59 years, and with a median of 8 days of intubation at the start of the study. The median PEEP was 3 cm H\textsubscript{2}O (range 0-10) and the median F\textsubscript{1\textsubscript{O}}\textsubscript{2}, was 0.45 (range 0.30-0.80). All patients had a physician’s order for chest physiotherapy at 4- or 6-hour intervals. Further information on individual patients appears in Table 1.

All data were collected by the same respiratory therapist (MTW), and patients were included in the study according to the work assignments of that therapist. No patients were excluded from possible inclusion in the study, beyond the requirements for intubation, mechanical ventilation, chest physiotherapy, and the availability of the study therapist.

For two consecutive chest physiotherapy treatments, the patients were suctioned with a Ballard closed-circuit suction catheter during one treatment, and were crossed over to suctioning with a conventional suction catheter (Pharmaseal, Valencia CA) during the other treatment. The order in which the catheters were used was randomly assigned. Both treatments were performed during the same day shift. Both the Ballard and the conventional catheter were size 14-Fr. The same suction pressure (120 torr) was used in all suction attempts with both catheters. Saline lavage was not used during suctioning with either catheter. We made no effort to control the suctioning of the patients at times other than during the chest physiotherapy treatments; patients were suctioned throughout the day by the nursing and respiratory therapy staff as clinically indicated.

All sputum obtained during each treatment was collected in a Luken’s trap, and the mass of the secretions was determined using an O-Haus Cent-O-Gram balance (Carolina Biological Supply Co, Burlington NC). At the completion of suctioning, the catheters were rinsed with a known volume of saline. To produce comparability of methodology, the Ballard catheters were disconnected from the airway and rinsed by the same method as that used to rinse the conventional catheters. The mass of the secretions was determined after correction for the mass of the Luken’s trap and the mass of the rinse solution.

We made 28 comparisons of the Ballard and the standard catheter in the 25 patients.

**Statistical Analysis**

Due to the non-normal distribution of the data, nonparametric statistical analyses were used; these were median, range, and the Wilcoxon matched-pairs test. A level of \( p \leq 0.05 \) was considered significant. Standard statistical methodology was used, and all statistical analyses were conducted using commercially available software (SPSS/PC+, SPSS Inc, Chicago IL).

**Results**

Findings for the individual patients are listed in Table 1. There was no significant difference between the quantity of secretions removed with the Ballard closed-circuit catheter (median 1.7 g) and the quantity removed with the conventional catheter (median 1.9 g) \( (p=0.88) \). In 15 instances, more secretions were removed with the Ballard catheter; in 13 instances, more were removed with the conventional catheter.
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<th>Ventilator Mode</th>
<th>Ventilation (L/min)</th>
<th>Fio2</th>
<th>PEEP (cm H2O)</th>
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Days of Intubation = number of days patient had been intubated at start of study.
COPD = chronic obstructive pulmonary disease.
CVA = cerebral vascular accident.
AA = aortic aneurysm.
MI = myocardial infarction.
OHS = open heart surgery.
A/C = assist/control.
SIMV = synchronous intermittent mandatory ventilation.
PS = pressure support.
Discussion

The results of this study suggest that the mass of secretions removed during suctioning is not affected by the use of a Ballard catheter versus the use of a conventional catheter. This is in contrast to the prior bias of therapists and nurses who worked in our ICU. Although other investigators have expressed concern regarding the effectiveness of the Ballard, we know of no other studies that have evaluated its ability to remove secretions.

Although we did not control or quantify the number of suction attempts with each catheter, it was the feeling of the therapist who collected the data that there was no apparent difference between the catheters in this regard. We also did not measure the thickness of secretions, but it was our clinical impression that this did not vary between treatments. The effects of confounding variables such as these are minimized due to the randomized crossover nature of our study design.

We believe that the Ballard is useful in patients requiring high FIO₂ and high PEEP levels. We have also found that the Ballard is useful in patients who are hemodynamically unstable, and in patients with elevated intracranial pressures. The Ballard may be useful to prevent contamination of the environment when patients are removed from the ventilator for suctioning, but we have not used it for this reason.

There are several reports in the literature that evaluate aspects other than secretion removal with the Ballard (Table 2). Use of the aperture of the swivel adapter for suctioning without removal of patients from the ventilator has also been reported. However, we no longer use this method in our hospital due to concerns regarding contamination of the catheter as it passes through the swivel adapter. The use of a side-hole endotracheal tube adapter for suctioning without removing the patient from the ventilator has also been described, but we do not believe that this method is commonly used.

This was a short-term study; further work is needed to evaluate secretion removal with long-term use of the Ballard, and with other designs of conventional catheters. Although additional work is also needed to evaluate closed-system suction catheters other than the Ballard, an abstract by Doorley et al indicated that the Ballard and Concord catheters (Concord Labs, Keene NH) were both clinically acceptable. An evaluation of the cost-

Table 2. Summary of Published Studies That Have Evaluated Aspects of the Use of the Ballard Suction Catheter

<table>
<thead>
<tr>
<th>Authors</th>
<th>Aspect Evaluated</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Craig et al</td>
<td>Arterial oxygenation</td>
<td>With preoxygenation, desaturation did not occur with either A/C or IMV. With no preoxygenation, desaturation occurred in patients on A/C, but not in patients on IMV when Ballard catheter used.</td>
</tr>
<tr>
<td>Carlton et al</td>
<td>Arterial oxygenation</td>
<td>No clinically important difference between Ballard catheter and conventional methods, even in patients receiving &gt; 10 cm H₂O PEEP.</td>
</tr>
<tr>
<td>Clark et al</td>
<td>Mixed-venous oxygenation</td>
<td>Higher mixed-venous oxygenation in patients suctioned with Ballard catheter.</td>
</tr>
<tr>
<td>Ritz et al</td>
<td>Airway contamination</td>
<td>No clear indication of either increased or decreased risk to patient from use of Ballard catheter for a period of 24 hours.</td>
</tr>
<tr>
<td>Deppe et al</td>
<td>Airway contamination</td>
<td>Ballard catheter increased the incidence of airway colonization, but did not increase incidence of nosocomial pneumonia.</td>
</tr>
</tbody>
</table>

A/C = assist/control ventilation.
IMV = intermittent mandatory ventilation.
effectiveness of the Ballard catheter also needs to be done.

Conclusions

The results of this short-term study suggest that the Ballard removes secretions as effectively as a conventional catheter. Further work is needed to evaluate the effectiveness of the Ballard during prolonged use. Further work is also needed to compare the cost of using the Ballard versus the cost of using a conventional catheter.

REFERENCES

Testing Airway Management Skills: Interactive Video Courseware vs ACLS Instructor

Arthur J Rubens DrPH

BACKGROUND: Lectures and demonstrations have been the teaching and testing strategies most often employed by the American Heart Association in Advanced Cardiac Life Support (ACLS) training. I compared the abilities of interactive videodisc (IVD) courseware and ACLS instructors to evaluate airway management skills. METHODS & MATERIALS: Twenty-two subjects were simultaneously tested during 30 attempts at endotracheal (ET) intubation and 34 attempts at esophageal obturator airway or esophageal gastric tube airway (EOA/EGTA) insertion. The instructors were blind to the visual and auditory messages produced. RESULTS: The IVD program and the ACLS instructors showed high agreement in their evaluation of student performance for time of intubation (95.5% ET; 100% EOA/EGTA), proper tube placement (91% ET; 93% EOA/EGTA), appropriate tube assessment (95.5% ET; 100% EOA/EGTA), and correct EOA/EGTA cuff inflation (100%). Lower levels of agreement were noted with ET and EOA/EGTA appropriate head positioning, and the evaluation of tooth pressure with ET intubation (60.5%, 76.5%, and 66.0%, respectively). The IVD system was unable to detect certain procedural errors associated with appropriate intubation procedure—syringe attachment, syringe removal after cuff inflation, and control of tube after intubation. The low agreement for tooth pressure suggests that the sensor-equipped manikin may better evaluate tooth pressure than does the observer. CONCLUSIONS: Although the IVD system shows promise as an adjunct method for instruction and testing, it cannot be considered suitable for ‘stand-alone’ instruction. Further research is needed to explore costs, skills retention, and possible impact of the medium for training hospital and prehospital-care personnel. (Respir Care 1991;36:849-856.)

Introduction

The predominate teaching strategy employed to convey and test the knowledge and psychomotor skills for Advanced Cardiac Life Support (ACLS) combines lecture and demonstration over 2 consecutive 8-hour days. The American Heart Association (AHA) in the 1986 JAMA Supplement1 states that the Association believes that this format markedly limits learning and retention. Over the past decade, an alternative method for instructing and testing ACLS has been developed that combines the use of computer and videodisc to produce what is called interactive videodisc (IVD) technology.

In this study, I sought to compare results of evaluation of airway management skills by Actronic’s Airway Management IVD course-
ware* with results by the traditional method of testing by the observations of ACLS instructors.

Background

ACLS Training

In 1975, the AHA developed specific standards for instruction and subsequent certification in ACLS. Instruction is by lecture and teaching stations at which the ACLS skills are demonstrated, practiced, and then tested. Included among the essential ACLS skills are endotracheal (ET) intubation and esophageal obturator airway (EOA) or esophageal gastric tube airway (EGTA) insertion. (The EGTA is a modification of the EOA, with the distal tip open to allow the passage of a gastric tube for decompression.)

ET intubation is a psychomotor skill that can be relatively easily acquired, and is a reliable instrument to ensure a patent airway, provide ventilation and oxygenation, and prevent aspiration. ET intubation is regarded as the optimal method for securing and protecting the airway in the compromised patient. The esophageal obturator airway developed by Don Michael in 1968 is described as requiring minimal instruction to teach its insertion, and minimal operator skill and dexterity, and may be inserted blindly by the rescuer. The AHA regards both ET intubation and EOA/EGTA insertion as standard ACLS procedures for airway management.

Computer-Assisted and Interactive Videodisc Instruction

In the early 70s, the laser-disc technology of IVD appeared and began to be used in the field of education. Advantages of the application of videodisc technology to instruction have been reviewed in this journal and elsewhere, and include the ability to hold quantities of visual (54,000 frames on a side) and digital information (10 million bits of information) and programming capabilities. IVD instruction has the advantage of providing immediate, personalized feedback and remediation where necessary, with the level of information difficulty controlled to match the pace of the student.

Since 1982, IVD technology has been used to teach the cognitive knowledge and practical skills of cardiopulmonary resuscitation (CPR). Studies of the IVD CPR program (and Lyness AL, unpublished data, 1985) have shown that students may learn the techniques in one third the time required by the traditional method.

Methods and Materials

The basic design of this pilot study followed the tenet of a quasi-experimental research format, with the data being collected prospectively. The study employed a simple one-group posttest-only design. This design, sometimes called a one-shot case study, was selected for convenience and because the testing format for ACLS airway management allows the same group to serve both as treatment and comparison group. The research was conducted at the Center for Emergency Medicine in Pittsburgh, Pennsylvania. A preliminary study was completed in September 1988, and the data were collected from October to December of that year. Fifteen of the 24 subjects were volunteers from an ACLS course. The other 9 subjects were students in a paramedic training class at the Center. All attempts at ET intubation and EOA/EGTA insertion made during the testing periods were included—30 attempts at ET intubation and 34 attempts at EOA/EGTA insertion.

Twenty two of the 24 study participants completed the pretest questionnaire that elicited demographic information, previous experience with airway management, relevant certifications, and their previous instructional exposures (Table 1). The study group was almost equally divided among health professionals in medicine, nursing, and paramed training, with most students having had previous experience with EOA/EGTA insertion and ET intubation with either manikin or patient.

The ACLS IVD airway management course has a video screen, for both the moving and still videodisc images, and a computer screen, which provides the options (user-interface menu) available to the student. An optical laser videodisc player is combined with an Apple II microcomputer. The audio system includes a 180-minute tape with 4

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*Suppliers are identified in the Product Sources section at the end of the text.
tracks, 3 with audio recordings and 1 with computer codes. The system is controlled by a software program supplied on diskette and is interfaced with an intubation manikin head equipped with sensors (Laerdal). Students use a key pad or a light pen to progress through the program, but during the pilot program only the practice and test menus of the program were used.

All students prior to being tested with the IVD system received (1) printed material outlining airway management and intubation,16 (2) a lecture addressing airway management and intubation, (3) laboratory practice using the sensor-equipped manikin supervised by the researcher and an ACLS instructor who provided remediation as necessary, and (4) a demonstration of the use of the IVD system.

The IVD testing station instruction occurred on Day 2 for the ACLS course participants, and directly after the conventional teaching station for the paramedic subjects. Two ACLS instructors who had no contact with each other during this testing period evaluated the students. The researcher was present throughout this testing to provide assistance with operating the selection menu of the learning system.

During the testing, the instructor was positioned so that he could not see the monitor but could see the student and the student’s manipulation of the devices and the intubation model. The remedial instructions from the IVD system were suspended during the testing program; however, the audio

---

### Table 1. Responses to Pretest Demographic Questionnaire (22/24 Participants Responding)

<table>
<thead>
<tr>
<th>Question</th>
<th>Number Responding</th>
<th>% of Responders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Education?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undergraduate</td>
<td>12</td>
<td>54.5</td>
</tr>
<tr>
<td>Graduate</td>
<td>10</td>
<td>45.5</td>
</tr>
<tr>
<td><strong>Specialty?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medicine</td>
<td>7</td>
<td>31.8</td>
</tr>
<tr>
<td>Nursing</td>
<td>5</td>
<td>22.7</td>
</tr>
<tr>
<td>Certified Paramedical Technician</td>
<td>2</td>
<td>9.1</td>
</tr>
<tr>
<td>Paramedical student</td>
<td>8</td>
<td>36.4</td>
</tr>
<tr>
<td><strong>Reason for Taking Course?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Job requirement</td>
<td>6</td>
<td>27.3</td>
</tr>
<tr>
<td>Recertification</td>
<td>7</td>
<td>31.8</td>
</tr>
<tr>
<td>General health education</td>
<td>1</td>
<td>4.5</td>
</tr>
<tr>
<td>Major study</td>
<td>8</td>
<td>36.4</td>
</tr>
<tr>
<td><strong>Previous EOA-EGTA Experience?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manikin</td>
<td>15</td>
<td>68.2</td>
</tr>
<tr>
<td>Manikin &amp; patient</td>
<td>3</td>
<td>13.6</td>
</tr>
<tr>
<td><strong>Previous ET Experience?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manikin</td>
<td>15</td>
<td>68.2</td>
</tr>
<tr>
<td>Manikin &amp; patient</td>
<td>5</td>
<td>22.7</td>
</tr>
<tr>
<td><strong>Previous Relevant Certifications?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACLS</td>
<td>8</td>
<td>36.4</td>
</tr>
<tr>
<td>BCLS</td>
<td>2</td>
<td>9.1</td>
</tr>
<tr>
<td>BTLS</td>
<td>1</td>
<td>4.5</td>
</tr>
<tr>
<td><strong>Previous Instructional Exposure?</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lecture</td>
<td>22</td>
<td>100.0</td>
</tr>
<tr>
<td>Film</td>
<td>18</td>
<td>81.8</td>
</tr>
<tr>
<td>Videotape</td>
<td>17</td>
<td>77.3</td>
</tr>
<tr>
<td>Interactive videodisc</td>
<td>1</td>
<td>4.5</td>
</tr>
</tbody>
</table>

---
recording of breath sounds to determine tube 
placement continued.

The students were graded simultaneously by the 
IVD system and the instructor. The instructor used 
AHA skills sheets to record student performance, 
with students receiving either a PASS or FAIL for 
their performance of the various skills of airway 
management. If the instructor concluded that the 
student’s first performance was unsatisfactory, the 
student could make two more attempts to perform 
satisfactorily. The IVD system presents a result 
screen at the conclusion of the student’s testing, 
which reports the results (PASS or FAIL) for each 
specific facet of performance. An overall assessment 
(PASS or FAIL) is indicated at the bottom of the 
result screen for performance area (ET and EOA/ 
EGTA). A tally of each attempt was noted by the 
researcher and the instructor. Students who correctly 
assess a right main-stem intubation for ET intubation 
are automatically prompted to pull back the tube 
and re-evaluate placement. Students who work 
independently on the system can have their results 
recorded in the test-bank memory if they choose.

Data Collection and Analysis

The results of the IVD-system and instructor 
evaluations were entered into a microcomputer 
utilizing a data management software package, and 
the numerical data were analyzed and interpreted 
by commercial software. Concordance for this study 
is defined as “when the learning system and the 
instructor agree” (ie, IVD system passed the student, 
and the instructor passed the student; or the system 
failed the student, and the instructor failed the 
student). Discordance is “when the system and 
instructor disagree on the student’s performance” 
(ie, IVD system passed the student, but the instructor 
failed the student; or IVD system failed the student, 
but the instructor passed the student). Essential 
facets of satisfactory performance for ET intubation 
and EOA/EGTA insertion were evaluated (see 
Appendix). Additionally, the estimated differences 
in the respective proportions for concordance and 
discordance data are reported, with a 95% 
confidence interval calculated.17

The consistency between the two learning 
modalities was also assessed by calculating percent 
agreement (Pa) and the respective value of Cohen’s 
kappa coefficient (K). Pa calculates the total 
proportion of consistent agreement for the two forms 
of evaluation:

\[
P_a = \frac{\text{concordance observations}}{\text{total number of observations}}
\]

K removes from the Pa the chance effect expected 
to occur and estimates the consistency between the 
two procedures.18 For most purposes, K values 
greater than 0.75 may be taken to represent excellent 
agreement beyond chance and values below 
approximately 0.40 may be taken to represent poor 
agreement beyond chance. Values between 0.40 and 
0.75 are generally taken to represent fair-to-good 
agreement beyond chance.19 In addition, data were 
obtained correlating the participant’s training 
(physician, nurse, paramedic) and previous expe-

<table>
<thead>
<tr>
<th>Step</th>
<th>n</th>
<th>Concordance</th>
<th>Discordance</th>
<th>Missing Data</th>
<th>Estimated Difference</th>
<th>Difference in Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Time for intubation</td>
<td>30</td>
<td>24</td>
<td>80.0</td>
<td>3</td>
<td>10.0</td>
<td>3</td>
</tr>
<tr>
<td>Sniffing position</td>
<td>30</td>
<td>20</td>
<td>66.7</td>
<td>9</td>
<td>30.0</td>
<td>1</td>
</tr>
<tr>
<td>Tooth pressure</td>
<td>30</td>
<td>18</td>
<td>60.0</td>
<td>10</td>
<td>33.3</td>
<td>2</td>
</tr>
<tr>
<td>Tube placement</td>
<td>31</td>
<td>27</td>
<td>87.1</td>
<td>2</td>
<td>6.5</td>
<td>2</td>
</tr>
<tr>
<td>Tube assessment</td>
<td>31</td>
<td>28</td>
<td>90.3</td>
<td>1</td>
<td>3.2</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 2. Comparison of Proportion with PASS/FAIL Probability for Endotracheal Intubation
Table 3. Comparison of Proportion with pass/fail Probability for EOA/EGTA Insertion

<table>
<thead>
<tr>
<th>Step</th>
<th>n</th>
<th>Concordance</th>
<th>Discordance</th>
<th>Missing Data</th>
<th>Estimated Difference</th>
<th>Difference in Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Time for intubation</td>
<td>34</td>
<td>31</td>
<td>91.2</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Jaw forward</td>
<td>34</td>
<td>26</td>
<td>76.5</td>
<td>8</td>
<td>23.5</td>
<td>0</td>
</tr>
<tr>
<td>Inflate balloon</td>
<td>34</td>
<td>28</td>
<td>82.4</td>
<td>2</td>
<td>5.9</td>
<td>4</td>
</tr>
<tr>
<td>Tube placement</td>
<td>34</td>
<td>23</td>
<td>67.6</td>
<td>0</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Tube assessment</td>
<td>34</td>
<td>23</td>
<td>67.6</td>
<td>0</td>
<td>0</td>
<td>11</td>
</tr>
</tbody>
</table>

Results

Results are presented in Tables 2 and 3 and establish the level of concordance between IVD and instructor evaluation of student performance. However, a 32% incidence of software malfunction (reported as missing data in Table 3) was recorded in EOA/EGTA tube placement and assessment. This primary malfunction involved air escaping past the stomach sensor and being interpreted as the student's performing ET intubation when, in fact, he was appropriately inserting the EOA/EGTA. Other similar malfunctions reported as missing data are seen in Tables 2 and 3. This high frequency of malfunction distorts the statistical significance for these procedures.

The estimated difference between the proportions was 87% for Tube Assessment and for ET intubation, 53% for Jaw Forward Position and 91% for Time for Intubation for EOA/EGTA intubation. The 95% CI for the difference in the proportions demonstrated a relatively wide interval with the calculated proportions.

The $P_a$ and $K$ values reported in Tables 4 and 5 in general demonstrate a high level of agreement between the two testing methods. Lower levels of agreement were noted with head positioning (60.5% and 76.5%) and application of tooth pressure during ET intubation (66%). The $K$ values were excellent for five, fair for three, and poor for three facets of the procedure. $K$ values correlated with $P_a$.^{19}

Skills check sheets completed by instructors revealed that certain facets of performance requiring subjective assessment could not be evaluated by the IVD system. "Poor performance" related to such procedures would not immediately fail the

Table 4. Percent Agreement ($P_a$) and Cohen’s Kappa Coefficient ($K$) between IVD and Instructor for Endotracheal Intubation

<table>
<thead>
<tr>
<th></th>
<th>Time</th>
<th>Sniffing Position</th>
<th>Tooth Pressure</th>
<th>Tube Placement</th>
<th>Tube Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_a$</td>
<td>95.5</td>
<td>60.5</td>
<td>66.0</td>
<td>91.0</td>
<td>95.5</td>
</tr>
<tr>
<td>$K$</td>
<td>0.91</td>
<td>0.34</td>
<td>0.24</td>
<td>0.73</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Table 5. Percent Agreement ($P_a$) and Kappa Coefficient ($K$) between IVD and Instructor for EOA/EGTA Intubation

<table>
<thead>
<tr>
<th></th>
<th>Time</th>
<th>Jaw Forward Position</th>
<th>Inflate Balloon</th>
<th>Tube Placement</th>
<th>Tube Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_a$</td>
<td>100</td>
<td>76.5</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>$K$</td>
<td>1.0</td>
<td>0.54</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>
Table 6. Occurrence of Subjectively Determined Poor Performance Not Detected by IVD

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endotracheal intubation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equipment</td>
<td>8</td>
<td>26.7</td>
</tr>
<tr>
<td>Intubation procedure</td>
<td>9</td>
<td>30.0</td>
</tr>
<tr>
<td>Cuff inflation</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>Tube movement</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>EOA/EGTA (n = 34)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equipment</td>
<td>2</td>
<td>5.9</td>
</tr>
<tr>
<td>Intubation procedure</td>
<td>2</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Discussion

The need to quickly achieve airway control in the critically ill patient is clearly established. I believe this paper reports the first pilot study of IVD instruction as an alternative method to teach this critical skill.

The findings of this study indicate that the IVD system shows a high degree of agreement with instructor evaluation for those facets of performance that use concise, objective criteria for their measurement: time for ET intubation and EOA/EGTA insertion, EOA/EGTA cuff inflation, and initial tube placement and initial tube assessment for both ET and EOA/EGTA. Less agreement is seen for skills involving more subjective evaluation: observation of proper head positioning for ET intubation, EOA/EGTA insertion, and application of tooth pressure during ET intubation. Although these skills are important, inadequate performance would not necessarily lead to a failed intubation.

I hypothesize that this variance in agreement on subjective skills depends in part on the perception and orientation of the instructor observing the student's performance. Further, the IVD system with its sensor capabilities may better monitor the skills of proper head position and tooth pressure than can the instructor if sensor function has been validated.

Table 7. Overview of Subjects' Attempts Related to Training, Experience, and Certification

<table>
<thead>
<tr>
<th>Procedure</th>
<th>ET Tube Insertion Attempts (%)</th>
<th>EOA/EGTA Insertion Attempts (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 (1)</td>
<td>2 (3)</td>
</tr>
<tr>
<td></td>
<td>6 (86)</td>
<td></td>
</tr>
<tr>
<td>Training</td>
<td>5 (71)</td>
<td>2 (19)</td>
</tr>
<tr>
<td>Medicine</td>
<td>3 (60)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>Nursing</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Paramedical</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>EOA Experience</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>None</td>
<td>2 (100)</td>
<td>—</td>
</tr>
<tr>
<td>Manikin</td>
<td>9 (60)</td>
<td>6 (40)</td>
</tr>
<tr>
<td>Manikin and patient</td>
<td>3 (60)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>Certification</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>ACLS</td>
<td>3 (38)</td>
<td>5 (63)</td>
</tr>
<tr>
<td>BCLS</td>
<td>1 (50)</td>
<td>1 (50)</td>
</tr>
<tr>
<td>BTS</td>
<td>1 (100)</td>
<td>—</td>
</tr>
<tr>
<td>7 (88)</td>
<td></td>
<td>1 (13)</td>
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<tr>
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RESPIRATORY CARE • AUGUST '91 Vol 36 No 8
During this pilot study, numerous occurrences of invalid sensor readings and machine malfunction were reported. Throughout the testing session, a second machine was available. Of course, this would not be feasible or cost-effective for the educational institution using IVD as an alternative to the conventional method. Events such as this not only result in frustration for the students but also represent downtime for the learning system. One of the often cited advantages of this type of instruction is its round-the-clock accessibility, but this presumes dependable operation.

The sensors were not able to detect some poor procedures—assembling and checking equipment, using the correct procedure to intubate, attaching and removing the syringe for cuff inflation, and maintaining tube control to assure that no movement occurs. Throughout the pilot study, an instructor was present to correct and give remedial instruction. In general, students did not repeat errors after this instruction.

According to Bork, an IVD system is regarded as a "stand-alone" instructional system. In my subsequent research, I found a student preference for the presence of an instructor or facilitator during practice and learning sessions. The Center employs a full-time media specialist to troubleshoot and to assist students with the system.

Not every student responds favorably to this instructional method. Auckerman and Shehee observed that certain students actively dislike being instructed and graded by a computerized system. Some of this negative reaction may be due to machine malfunction (even though malfunctions are quickly corrected). However, some dislike appears to result from improper student interaction with the system messages. For example, although told by the system to ventilate the manikin at the sound of the beep, the student begins immediately. It has been reported that after initial frustration, recertifiers respond favorably. I have observed that students already familiar with the system move more rapidly through the learning and testing process. In 1982, Hon predicted that learning time for CPR could be reduced from 4 hours for initial certification to ½ hour for 6 months and 1 hour for 1-year recertification.

In January 1989, the American Heart Association accepted the Actronics Interactive Videodisc Airway Management course as a valid alternative for ACLS certification. The cost of the system (hardware and software) and an annual service contract is approximately $30,000.

The purpose of my study was to determine whether the system was indeed valid for ACLS training. The results support the use of the system, but also reveal limitations of the system and of this form of instruction and suggest future research. I have observed in subsequent use that many of the malfunctions encountered in the pilot study have been eliminated, but evaluation of downtime and maintenance needs to be conducted.

Conclusions

The IVD learning system showed high agreement with instructor observation in certain facets of airway management; however, the system was unable to detect certain errors related to what has been labeled poor procedure. Additional research is needed to explore costs, learning effectiveness and retention, and possible impact of the medium on training in-hospital and prehospital caregivers.

ACKNOWLEDGMENTS

I thank Actronics Inc for providing the software and equipment for my research and Mr Walt Stoy and his staff at the Center for Emergency Medicine of Western Pennsylvania, Pittsburgh, for their participation.

PRODUCT SOURCES

System distributor:
Actronics Inc, Pittsburgh PA

Software components:
Software: Airway Management, Version 2.0 (two 5 1/2" disks) Actronics Inc, Pittsburgh PA
Videodisc: Airway Management, Version 2.0, Actronics Inc, Pittsburgh PA
Audiotape: Airway Management, Version 1.0, Actronics Inc, Pittsburgh PA

Hardware components:
Computer: Apple II-E customized with interface cards
Apple Computer, Cupertino CA
Videodisc: Panasonic videodisc player, Elgin IL
Manikin: Laerdal Intubation head, sensor-equipped by Actronics Inc, Pittsburgh PA

Software for data acquisition and analysis:
dBASEIII Plus, Ashton-Tate, Torrance CA
SPSS/PC, Version 2.0, SPSS Inc, Chicago IL
REFERENCES


APPENDIX

Instructor Checklist for Procedure Performance

Endotracheal Intubation

1. Time: Time for intubation (> 30 seconds = F)?
2. Sniffing Position: Manikin's head placed correctly in the sniffing position for intubation procedure?
3. Tooth Pressure: Unnecessary pressure exerted on teeth during procedure?
4. Initial Tube-Placement: ET tube initially placed correctly in trachea?
5. Tube Assessment: Tube location correctly assessed?
6. Right Main-Stem: Tube incorrectly inserted into right main-stem? Student correctly detected error and corrected it?
7. Checking Equipment: Presence and integrity of intubation equipment checked correctly?
8. Intubation Procedure: Laryngoscope held correctly in left hand, and inserted correctly into manikin's mouth, moving tongue from right to left?
9. Cuff Inflation: ET cuff inflated with 4-6 mL of air, and syringe removed after inflation?
10. Moving/Releasing Tube: Tube not released or moved after insertion through vocal cords?
11. Final Results: PASS/FAIL judgment for each attempt.

EOA/EGTA

1. Time: Time for intubation (> 30 seconds = F)?
2. Jaw Forward Position: Patient correctly placed in neutral position with jaw forward?
3. Initial Tube-Placement: EOA/EGTA tube initially placed correctly in esophagus?
4. Tube Assessment: Tube location correctly assessed?
5. Checking Equipment: Presence and integrity of EOA/EGTA intubation equipment correctly checked?
6. Correct Intubation Procedure: Jaw grasped correctly, and tube with mask attached inserted following curvature of the pharynx until mask seals firmly over mouth and nose?
7. Final Results: PASS/FAIL judgment for each attempt.
State Licensing Boards and Your Legal Rights

Harold R Rauzi JD RRT

Background and Introduction

A decade ago, only one state licensed respiratory care practitioners. Today, credentialing laws have been passed in 30 states and Puerto Rico,\(^1,2\) and efforts to pass such legislation continue in other states.\(^1-3\) Practitioners may find it helpful to develop a basic understanding of their legal rights as those rights apply to the activities of state licensing agencies.

The modern scheme of licensing health care professionals began in 1873 when Texas enacted the first mandatory licensing of physicians. The first mandatory registration of nurses was enacted in 1938.\(^4\) (Of course, occupational licensing is not limited to health care—the typical state licenses 30 occupations.\(^5\))

Alternate forms of state regulation of occupations and professions include certification and title protection. Under such systems, only those who have met the qualifications set by law and state board regulation may use the title Registered or Certified. Unlike licensing, certification or title protection acts do not prohibit uncredentialed persons from practicing but merely restrict the use of the official titles.\(^4\) Eleven states have chosen this option for credentialing respiratory care personnel.\(^1\)

Your professional license represents a valuable property right; however, there is no absolute right to follow a given profession.\(^6\) Over a century ago, in the landmark case of *Dent v West Virginia*,\(^7\) the Supreme Court recognized that the state's obligation to protect the public from incompetent practitioners outweighed the individual's right to pursue a particular profession. Consequently, the Court held that states may require applicants for licensing to pass an examination and fulfill continuing education requirements.\(^7\) A few years after *Dent v West Virginia*, the Supreme Court approved licensing laws that require applicants to be "of good moral character."\(^8\) A state's licensing power does not, however, completely outweigh the individual's right to practice. An applicant cannot be denied a professional license, nor may a practitioner's license be suspended or revoked without "due process of law."\(^9,10\) A practitioner is entitled to due process when applying for a license and when facing disciplinary action. The licensing agency is also bound by the rule of due process when promulgating rules and regulations that affect the manner in which individuals practice their profession.\(^4,6\)

State licensing boards have only as much authority as the legislature grants in the act creating the board.\(^6,10\) Additionally, in many states, an administrative procedure act sets forth the procedures that the respective state agencies must follow when making rules or investigating and adjudicating disputes.\(^6,8,10\)

Regulations Associated with Licensure

Entry to the Profession

Usually a licensing act establishes minimum educational requirements for entry into the profession. The licensing boards, by regulation, fill in the details.\(^4,6\) For example, frequently a statute stipulates that an applicant must be the graduate of an "approved school." The board is empowered to designate which schools are approved. As another example, a statute may require that applicants pass an examination. The board is responsible for

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designing the test and determining the passing score.\textsuperscript{11}

Legislation creating respiratory care boards also limits the authority of the boards. The board must remain within the boundaries of the enabling act. If your state’s licensing act requires only that respiratory care practitioners be graduates of a school approved by the Joint Review Committee for Respiratory Therapy Education, the board cannot enforce a regulation requiring that the applicant also have a master’s degree.\textsuperscript{4}

The educational and experiential requirements for licensing must be “rationally related” to the applicant’s capacity to practice the profession.\textsuperscript{12} For example, pulmonary physiology is rationally related to respiratory care; therefore, a respiratory care board may make course work in pulmonary physiology a prerequisite to taking the respiratory care examination and/or include it as an examination subject. On the other hand, a question regarding the prima facie elements of a cause of action sounding in products liability would be inappropriate on the respiratory care exam (but would be appropriate on the bar exam).

Licensing boards (and courts) have had trouble determining what constitutes good moral character for purposes of professional licensure. In the past under “good moral character” provisions, homosexuality automatically disqualified one from professional licensing.\textsuperscript{5} Similarly, applicants have been denied licenses because they were living, unmarried, with someone of the opposite sex.\textsuperscript{12} Today, private, noncommercial sex acts between consenting adults are, in the legal sense, irrelevant to one’s fitness to practice a chosen profession.\textsuperscript{13} However, licensing boards may deny a license to persons who have been convicted of felonies or of misdemeanors involving moral turpitude (eg, fraud and theft).\textsuperscript{9,14}

Nevertheless, when a state licensing board follows the proper rule-making procedure and adopts regulations within the scope of its authority, those regulations have the force of law. Courts will uphold rules that require that an applicant fill out the proper form completely, file the application on time, submit grade transcripts, and pay the appropriate fee.

Disciplinary Procedures

In general, licensing acts specify the grounds for disciplinary actions. Courts have upheld disciplinary actions for such things as criminal convictions, performing acts beyond the scope of practice, incompetency, and drug impairment.\textsuperscript{9,15-19}

Of all areas of administrative law, it is in the area of disciplinary proceedings that the concept of due process has been most fully developed. A practitioner who is facing discipline by a state board is entitled to:

- a reasonably definite statement of the charges against him/her;
- a fair hearing before an impartial board;
- advance notice of the time and place of the hearing;
- the right to present evidence and witnesses in his/her favor; and
- the right to cross-examine the witnesses against him/her.\textsuperscript{5,6}

The courts will not uphold disciplinary actions based on vague charges, such as “conduct unbecoming an R.T.”’ The law requires a more specific charge, such as “falsified charting” or “drinking on duty.”

In some states, the law does allow the board to suspend a practitioner’s license without a hearing if allowing the accused person to continue practicing would endanger patients. However, a formal hearing, meeting all of the standards listed above, must be held as soon as practicable.

In most states, the practitioner has the right to legal counsel during administrative disciplinary proceedings.\textsuperscript{5,9} (Although one has the right to legal counsel, the state is not required to provide an attorney. That principle only applies to criminal charges. In this case, the practitioner’s right to counsel simply means that if one chooses to retain an attorney, the board must permit him/her to represent the practitioner.)

In general, a licensing board has several options should it choose to discipline a practitioner, ranging from reprimand through probation, restrictions on practice and suspension, to revocation of license.\textsuperscript{4,6,9}

Along with the power to discipline, licensing boards usually have the authority to conduct investigations, including the power to issue
subpoenas and the authority to take sworn testimony.10,20 Often such investigations are conducted under the aegis of the state attorney general’s office or a similar agency.

Although an anonymous tip is usually sufficient grounds to begin an investigation, such a tip would be insufficient to justify disciplinary action. As already noted, the practitioner facing sanctions has the right to cross-examine his/her accuser.

Agency (board) hearings are less formal than court proceedings. Nevertheless, (in most states) at a disciplinary hearing, witnesses must testify under oath. Moreover, the board must act in an impartial and unbiased manner.5,6 Disciplinary actions by licensing boards are subject to judicial review. However, there are significant limitations on court involvement.

Most states follow the rule requiring “exhaustion of administrative remedies.”16 This means that the practitioner must first participate in the administrative process before turning to the courts. For example, in many states, a disciplinary hearing is conducted before a hearing officer (sometimes called an administrative law judge). The hearing officer (often an attorney) then makes a report to the board recommending a course of action (eg, to dismiss the complaint or to suspend the practitioner for a period of time). The board may accept, reject, or modify the hearing officer’s recommendation. Suppose that a hearing officer recommends that a practitioner’s license be suspended, but, before the hearing by the full board, the practitioner files an action in court against the board. The court will refuse to consider the case. The practitioner must participate in the board hearing and only then, if the board’s ruling is adverse, he/she may turn to the courts for relief.20

The extent to which the courts will review a board decision varies by state. In most states, the court will review only the fairness of the investigation and hearing. If the court believes the board acted arbitrarily, the court will order the board to hold a new hearing. The court will not second guess the board’s decision but will only evaluate the fairness of the hearing process.9,20 However, in a few states, the courts will conduct a trial de novo, essentially starting from scratch, taking testimony anew from all the witnesses, and reaching its own decision as to whether the practitioner should be disciplined. (The requirement that the practitioner exhaust administrative remedies before turning to the courts also applies when a practitioner’s application for a license is denied. The rejected applicant must first follow the board’s procedure for reconsideration before filing suit.)

By tradition, the regulation of occupations and professions has been a function of state government. Recently, the federal government has begun to adopt laws that affect individual health care professionals. Directly related to licensing and disciplinary procedures is the Health Care Quality Improvement Act of 1986 (HCQIA).21 The purpose of this act is to encourage hospitals and other health care organizations to establish and maintain active professional peer review committees. This statute is primarily concerned with restriction, suspension, or revocation of medical staff privileges.21,22 It has little direct bearing on respiratory care practitioners, nurses, or other therapists and technicians who are generally classified as hospital employees rather than medical staff. However, two points of which nonmedical staff personnel ought to be aware are:

- HCQIA requires in-house, professional peer review committees to report disciplinary action against physicians to a national registry, as well as to the state medical board. The law permits such committees to report incompetence or misconduct of any other licensed health care practitioner to the appropriate board.21
- HCQIA also shields professional peer review committee members and those who assist such committees (including witnesses) from liability for slander or defamation regarding those persons being investigated by the committee.21,22
- HCQIA extends due process protection to physicians who are subject to revocation, suspension, or restriction of hospital privileges as a result of a professional peer review action.21

In Conclusion

Because the laws vary, often significantly, from state to state, this discussion has of necessity been general. Nonetheless, there are a few broad principles common to all jurisdictions.
A license to practice a profession is a valuable property right. It is not, however, an absolute right. When a practitioner is denied a license or is faced with the possibility of having his/her license restricted, suspended, or revoked, the practitioner is entitled to the due process of law. It behooves you to be familiar with your state licensing act and your board rules and regulations. Most public libraries have the state statutes available.

Also, in most states, all rules and regulations adopted by the various state agencies are published in compilations (sometimes called the “Administrative Code” or “Official Register,” or some similar name), which are available at most larger public libraries. Your state board can provide you with copies of all rules and regulations it has adopted. (They may charge a small fee and/or refer you to a state printing office.)

If you are familiar with these rules and regulations, especially those regarding renewal and continuing education, you may never have cause for concern about your license. If you have any problem with your state board, ask (in writing) for a full explanation. If you do not receive a satisfactory answer promptly or at the first hint that you are facing disciplinary action, consult an attorney. Your license to practice is too valuable to risk its loss.

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15. Maniglia v. Department of Professional Regulation, 446 So. 2d 186 (Fla. App. 1983) (Physician’s license revoked for giving false information on license application).
Adrenergic Bronchodilators: Trends In Drug Design

Hugh S Mathewson MD

The molecular structures of adrenergic drugs have been well differentiated and specialized since the naturally occurring catecholamines were isolated and introduced into medicine nearly a century ago. After epinephrine was found to be a rapidly effective bronchodilator, the basic phenylethylamine structure was successively modified over the next several decades, so that today adrenergic agonists with high beta_2 specificity are available. Albuterol, terbutaline, pirbuterol, and bitolterol are approved in the United States; these agents, administered by aerosol, will usually reverse acute bronchoconstriction in a few minutes. Bronchodilatation is maximal in about 30 minutes and persists for about 4 hours.

Adrenergic drugs have pharmacologic actions that are important in asthma therapy in addition to their relaxant effect on bronchial smooth muscle. Beta_2 agonists increase mucociliary transport, inhibit the release of mediators, and suppress permeability edema. As rescue drugs for acute asthma attacks, they are unsurpassed. However, their duration of action is too short to diminish the tissue responses that follow the onset of the asthmatic episode. Although a 4-hour period of bronchodilatation is a considerable span, it is usually insufficient to cover the nocturnal sleep period. Also, there may be a rebound of bronchial hyperreactivity, attributable to the effects of eicosanoids and other bronchial spasmodens that outlast the period of action of the drug. In addition, currently available beta_2 agonists can cause muscle tremors and may elicit central-stimulant (amphetamine-like) effects. When given parenterally, they are likely to cause cardiovascular (beta_1) side actions.

The two goals in development of improved beta-adrenoceptor bronchodilators are increased functional selectivity and longer duration of action. These goals can be approached at least four ways through drug design: (1) by increasing the lipophilic nature of the molecule, (2) by providing functional groups that attach to exteroceptor sites, (3) by synthesizing agents in the form of prodrugs that are slowly bio transformed in vivo to active bronchodilators, and (4) by designing drugs that are effective topically but are rapidly inactivated when absorbed systemically, thus minimizing side effects.

**Lipophilic Potentiation**

The efficacy and duration of action of adrenergic stimulants can be augmented if the number and size of lipid-soluble groups in the molecule are increased, indicating that receptor binding is favored by lipophilicity. This can be accomplished by the addition of hydrocarbon-like groups or halogens. These principles are exemplified by the newer bronchodilator compounds, formoterol and tulobuterol (Fig. 1).

Formoterol, so named from its formamido substituent on the benzene ring, was first described in 1977. Its side chain has the tert-butyl terminus found in albuterol and terbutaline, but with an appended p-methoxyphenyl group, which increases lipid solubility. Several studies have demonstrated that formoterol retains much of its bronchodilator effect for 8-12 hours after aerosol administration. In a long-term study, its effectiveness was prolonged significantly more than albuterol, and it was definitely preferred by patients.

Tulobuterol has been compared with albuterol in blind crossover studies, both orally and by aerosol. By the oral route, tulobuterol was found to be significantly longer acting, requiring only twice daily dosage for maintenance. The side actions of the two drugs were comparable. By aerosol, tulobuterol was shown to be about equal to albuterol with respect to onset, peak, and duration of response.

**Exteroceptor Binding**

If the lipophilic side chain is considerably lengthened, there is evidence to show that adrenerceptor binding is enhanced through attachment of this appendage to a cellular site adjacent to the adrenerceptor. The duration of the beta_2 response is thus prolonged. Salmeterol (Fig. 1), a saligenin derivative similar to albuterol, has an 11-atom side chain extending from the amino group of the albuterol-like structure to a terminal phenyl group. It is postulated that this long side chain binds to an exteroceptor near the beta_2 receptor, and that this accounts for the prolonged action of the drug. A similar property has been suggested for the formoterol molecule, but the evidence is less convincing.
Salmeterol shows bronchial relaxant efficacy for at least 12 hours in experimental studies. Clinical trials have confirmed its sustained effect usually for periods exceeding 8 hours after administration by aerosol. Current reports indicate that salmeterol is in most respects equal or superior to albuterol as a bronchodilator, and the prolonged relief of asthmatic symptoms has resulted in noteworthy patient acceptance.

Prodrugs

These are compounds that are pharmacologically inactive but are slowly biotransformed, usually by ester hydrolysis, to active beta₂ agonists. An ideal steady-state concentration of the active metabolite is difficult to achieve. Too rapid hydrolysis exposes the patient to side actions. This was the experience with albuterol, the bis-isobutyric ester of terbutaline, which was hydrolyzed so promptly that the effects of albuterol were virtually indistinguishable from those of terbutaline. On the other hand, if hydrolysis is too slow a therapeutic level of the active metabolite may never be reached.

A more effective prodrug is bitolterol, the bis-p-toluic ester of crotol, a catecholamine derivative once used as a bronchodilator. Although hydrolysis of the prodrug is reasonably slow, the colterol produced is, like other catecholamines, rapidly biodegraded, and the bronchodilator effect is evanescent. Its use in the United States is limited to aerosol administration.

Bambuterol is another prodrug that yields terbutaline on ester hydrolysis. When given orally it is reported to sustain plasma therapeutic levels for 24 hours, considerably longer than the 5-6 hour period attained by single doses of terbutaline itself. Bambuterol was also found to be more effective than a slow-release preparation of terbutaline.

Drugs with Reduced Systemic Side Actions

Because adrenocceptor selectivity is limited, the side actions of beta₂ agonists are difficult to minimize. However, it may be possible to design compounds that are potent bronchodilators when applied topically to the bronchial mucosa and that are promptly biotransformed or eliminated when systemically absorbed. Such compounds have been referred to as “soft drugs.” An experimental ester derivative, ZK 90055 (Fig. 1), is representative of this type of agent. It is about 10 times more potent a bronchodilator than is isoproterenol, but the ester group is quickly hydrolyzed in the liver to the corresponding carboxylic acid, which is practically inactive. When given by aerosol, the drug that is swallowed is mostly metabolized on first pass through the liver. Whether the drug absorbed from the lung is just as rapidly hydrolyzed has not been determined.

As yet, researchers have been unsuccessful in abolishing muscle tremors and cardiovascular effects in new drug formulations. Two compounds (procaterol and broxaterol) are under investigation with this goal in mind. Recently two experimental compounds (OH 25 and D2343) were shown to combat bronchospasm in animal experiments without affecting skeletal muscle and the heart. Unfortunately, these desirable attributes were not borne out in clinical trials.

Beta₂ agonists in present use are rather ineffective in protecting the asthma-prone patient from provoking stimuli. Also, they appear not to alter bronchial hyperreactivity.
may even increase it.\textsuperscript{30} For optimum control of chronic asthma, general consensus is that aerosolized corticosteroids must be given concurrently to reduce inflammatory tissue responses. Supplemental use of cromolyn sodium or nedocromil may be advisable during periods of heavy pollen release. These requirements do not detract from the necessity for beta agonists when immediate treatment of bronchospasm is required.

**REFERENCES**

Increased Shortness of Breath, Purulent Sputum, and Fatigue in Patient with Spinal Arthritis

Digpal Chauhan MD

A 49-year-old Caucasian man was admitted to the hospital for increased shortness of breath, purulent sputum, and easy fatigability of several weeks duration. He had suffered from arthritis of the spine for many years and was taking indomethacin (Indocin) for it. He denied having hemoptysis, fever, chills, or night sweats. His history was positive for pneumothorax (20 years earlier) and negative for pansinusitis, nasal polyposis, and pancreatitis. He had a 30-pack-year smoking history but had quit smoking prior to this admission.

The patient weighed only 82 pounds and was extremely debilitated, appearing older than his stated age. Examination of the spine revealed markedly diminished range of motion. Optic fundi were normal. Cardiac examination revealed a systolic click. Examination of the lungs revealed bronchial breath sounds and crackles in the right upper lobe, with expiratory wheezes in both bases. Digital clubbing was not present. Genital examination was normal.

Sputum culture grew normal flora. 8 AM and 4 PM plasma cortisol levels were normal. Serum testosterone was normal. Alpha1-antitrypsin level and genotyping were normal. Thyroid functions, including serum T4 (thyroxine), T3 (triiodothyronine) by radio-immune assay, and thyroid stimulating hormone (TSH) were normal. Bronchial washings were negative for acid-fast bacilli (AFB) smears and cultures and fungal cultures and cytologies were...

Fig. 1. Posterior-anterior (PA) chest radiograph of a 49-year-old man with longstanding history of arthritis of the spine, admitted to the hospital for increased shortness of breath, purulent sputum, and fatigue.
also negative. Complement fixation and immunodiffusion titers were negative for Histoplasma and Coccidioides. The serum IgE level was normal, and serum precipitins for *Aspergillus fumigatus* were negative.

Pulmonary function testing revealed TLC of 56% predicted, FVC 1.328 L (29% predicted); FEV₁ 0.810 L, FEV₁/FVC ratio 61%, and D_LCO 98% predicted. Analysis of a sample of arterial blood taken with the patient breathing room air revealed pH 7.45, P_{aco₂} 29 torr, and P_{ao₂} 87 torr. Radiographs of the chest (PA and lateral) and of the spine and sacroiliac joints were obtained, and are shown in Figures 1-3.

**Questions**

**Radiographic Findings:** What abnormalities appear in the radiographs shown in Figures 1-3?  
**Diagnosis:** What conditions should be considered in the differential diagnosis? What is the likely diagnosis?  
**Further Actions:** What further diagnostic and therapeutic actions are indicated?

**Answers and Discussion on Next Page**
Answers

Radiographic Findings: The PA chest radiograph (Fig. 1) reveals bullae in both upper lobes. In the lateral chest radiograph (Fig. 2A), multiple curvilinear lines can be seen above the major fissure, which suggests that the bullae are above the major fissure (no bullae are seen over the cardiac shadow) and are located mostly in the upper lobes. The major fissure extends from the level of the fifth thoracic vertebra obliquely downwards to the diaphragm at a point just behind the anterior costophrenic angle, as illustrated in the schematic shown in Figure 2B. In Figure 2A, the upper arrow shows anterior spinal ligament calcification, and the lower arrow shows squaring of vertebral bodies and osteoporosis. The radiograph of the sacroiliac joints (Fig. 3) reveals fused sacroiliac articulations and obliteration of the joint space (shown by the arrow).

Diagnosis: The differential diagnosis includes bullous emphysema due to smoking or alpha-antitrypsin deficiency; chronic indolent infections, particularly mycobacterial or fungal infections, or sequelae of acute necrotizing pneumonia with pulmonary gangrene; cystic fibrosis; and fibrobulous disease secondary to end-stage pulmonary fibrosis due to rheumatoid lung, sarcoidosis, or silicosis. The correct diagnosis for this patient is ankylosing spondylitis and fibrobulous disease in the apices of both upper lobes. The radiographic findings of fused sacroiliac articulations, anterior spinal ligament calcification, spinal fusion, and increased squaring of the vertebral bodies (so-called ‘bamboo’ spine) are all diagnostic of ankylosing spondylitis.1,2

Further Actions: Another bronchoscopy may be indicated because secondary infection with Aspergillus fumigatus is common in this condition. The patient should be tested for the HLA B-27 antigen to confirm the diagnoses. Bronchodilator and antibiotic therapy and nutritional supplementation are indicated.

Bronchoscopy was repeated with negative microbiology studies and negative bronchial washings for cytology. Our patient was HLA B-27 antigen positive. Bronchodilator and antibiotic therapy, liquid-formula nutritional supplementation, and physical therapy were instituted.

Discussion

Our patient is doing well on this regimen; he has gained weight and has experienced an improvement in exercise tolerance. Periodic follow-up evaluation is scheduled for early identification and treatment of secondary opportunistic infections and aspergillosis. Infected cavities usually present with fever, purulent sputum, intermittent hemoptysis, and air-fluid level visible on chest radiograph.

Ankylosing spondylitis is a chronic inflammatory disease that primarily affects the axial skeleton, often resulting in progressive stiffening of the spine and sacroiliac joints. Clinically important pleuro-pulmonary manifestations are uncommon. In a review of ankylosing spondylitis patients from the Mayo Clinic by Rosenow,3 the incidence of pleuropulmonary manifestations was about 1.3%. HLA B-27 antigen is positive in 90-95% of Caucasian patients with ankylosing spondylitis compared to 6-10% of normal healthy persons.4 The majority of ankylosing spondylitis patients with upper-lobe fibrobulous disease are HLA B-27 positive.4 Pulmonary manifestations of ankylosing spondylitis generally fall into one of two categories: chest-wall restriction or upper-lobe fibrobulous disease.4

Chest-wall restriction in ankylosing spondylitis is the result of inflammatory disease of the thoracic spine and consequent fusion of costovertebral joints. Usually TLC and VC are mildly to moderately decreased.4 In general, the restrictive defect caused by a stiff spondylitic thorax is usually compensated for by increased diaphragmatic excursion.5

Upper-lobe fibrobulous disease is the most common pulmonary abnormality found in patients with ankylosing spondylitis.5 The etiology of fibrobulous disease is unknown. In most cases the disease begins in one upper lobe but eventually becomes bilateral. The early signs are apical pleural thickening and interstitial infiltrates in both apices; later the areas of interstitial fibrosis break down with subsequent cavity formation. The disease may steadily progress or may remain stable for years.4 In some cases the pleural thickening and fibrosis become extensive and result in upward retraction of one or both hila; upper-lobe bronchiectasis and changes indistinguishable from those found in pulmonary tuberculosis may also occur.4

These cavities have predilection for secondary infection by Aspergillus fumigatus and mycobacteria.
(especially atypical mycobacteria). It has been suggested that reduced ventilation to the apical portion of the lungs, due to rigidity of the thoracic spine, may play some role in the development of apical fibrosis and cavitation and subsequent complicating infections. Also, prior radiation therapy to the spine for ankylosing spondylitis may give rise to radiation fibrosis of the lungs.

Patients with ankylosing spondylitis may have other respiratory complications. Intubation may be particularly difficult in patients with ankylosis of the cervical spine, and may be accompanied by an increased risk of cervical fracture. In view of this risk, the use of a modified technique of awake intubation (utilizing fiberoptic bronchoscopy) has been advocated. Also, as in cases of rheumatoid arthritis, cricoarytenoid-joint ankylosis can occur and may result in hoarseness, sore throat, and vocal-cord dysfunction, requiring tracheostomy or arytenoidectomy.

Fibroblastic lung disease is usually asymptomatic, and no evidence exists that any form of therapy alters the progression of this disease provided the cavities are not infected. Thoracotomy is not usually undertaken to remove these lesions unless major hemoptysis has occurred because surgical procedures may be complicated by chronic empyemas and bronchopleural fistulas in as many as 50% of the cases.

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American Association for Respiratory Care
37th Annual Convention and Exhibition
Atlanta, GA • December 7-10, 1991

• Keynote Address
• Donald F. Egan Scientific Lecture
• Program Committee Special Lecture
• 24 Symposia
• Open Forum Papers
• Open Forum Minisymposia
• AARC Awards Ceremony
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Make plans to attend the best and largest respiratory care meeting in 1991!

"Paperwork is a tedious, time-consuming, nitpicky, counterproductive pain in the butt. Unfortunately, it is also one of the intrinsic evils of working in the healthcare professions. Its profound aggravation and utter vacuity sometimes obscure its genuinely useful purposes."

This astonishing declaration, opening Chapter 7 of Barton’s Pulmonary Rehabilitation/Homecare, has a sublimity of its very own that is positively thundering as it rolls and boils in the vastness of its existentialist scope and meaning. It says, I think, that useful purposes are to be found by piercing the veil of counterproductive, evil emptiness! (Goodness! Permit me a furtive glance over my shoulder as the lamps of enlightenment dim around me.)

Richard O. Barton BA RRT is a pulmonary rehabilitation teacher/consultant at the Pacific Presbyterian Medical Center, San Francisco CA. His book, in two parts, Preparation and The Program, is separated into 13 chapters. A fairly comprehensive Table of Contents is of some limited value in the absence of an index, footnotes, or references. The author states in his preface that the subject matter—that is, rehabilitation and home care—does not lend itself “to this method” (indexing, footnoting, and referencing). It appears that he unsuccessfully tried to fashion a coherent book out of largely anecdotal material heavily interspersed with personal bias and prejudice.

From the beginning it proved to be a very difficult volume to read. Nearly every page is a study in officiousness punctuated by malapropisms, mistaken assertions, imprecise terms, contradictions, and local idioms, not to mention grammatical errors coupled with unusual page layouts. Be that as it may, by the time I finished the first chapter, I was captivated! I read the work from cover to cover, not for any light it might shed on my own practice, but rather as an exercise in perseverance.

Mr Barton introduces, on Page 51, the notion of selecting and providing patients with handbooks. He discusses the need for and broad availability of such items, but unfortunately has no recommendations to make or titles to offer.

On Page 37, the reader is informed that social workers do not teach in the program only to learn on Page 43 that social work/discharge planning should be represented in the teaching process. Further, Barton chastises clinicians on Page 7 for inconsistencies in teaching rehabilitative techniques while subsequently pointing out on Page 10 that, "For this reason no two ... plans will be exactly the same at the individual level."

As mentioned, there are a number of malapropisms in Pulmonary Rehabilitation/Homecare. A representative example is found on Page 13, "To ask an intubated and heavily sedated patient, 'How are you doing today?' ... is not likely to illicit much in the way of clinically significant information." I can readily conjure up a number of shady venues for illicit information. I think, in this instance, though, that the author wishes to emphasize the futility of trying to elicit subjective data from the patient described. I am compelled to ask the question, But who would even try?

The author uses the noun "rote" throughout to modify another noun, "practice." The more correct phraseology is "repetitious practice" rather than "rote practice."

"Nutritional" should have been used on Page 37 instead of "nutrition." The past participle "known" is used in place of the verb "know" on Page 50. These are just two examples of grammatical errors.

Page 50 has a startling quote, "So then, prepare to dig into your old texts and brush up on P, D, and TISS, as well as FCC-DOT tanktop graffiti." This comment is in the context of a discussion about cylinder gases used in hospitals that were built before "indoor O2 plumbing" was available. Barton does a disservice in trivializing these important stamped safety markings on the shoulders of high-pressure cylinders.

I identified a number of false or misleading assertions in this paperback. He writes (Page 41), "Suppose a patient is to be sent home using continuous O2. The dwelling has gas appliances with open pilot lights, and this precludes the use of a tank (sic) or liquid system. Let’s further suppose that the patient’s financial assistance source will not cover a concentrator. What options remain?"

He not only fails to answer the very question he poses, but the question itself is based on a mistaken certainty. There need be a space of only 10-15 feet between a therapy oxygen source (including a concentrator) and an open flame or spark source.

We are instructed to garner the support of the medical director to convince the institution’s administration of the value of a rehabilitation program (Page 45). This instruction presupposes administrative resistance, even hostility, to such a notion. In fact, administration in its continual quest for "market share" is likely to be the very driving force behind actual program development.

On Page 69, we are told in no uncertain terms that inpatient care is a hospital’s primary function "by
definition.” This simply is not true, as the author should know from working in a major metropolitan region. In 1989, one large hospital in Texas had only 40,000 admissions while handling 582,700 emergency room and outpatient clinic visits.3

“Pulmonary disease, by definition, impairs breathing.” This writer on Page 123. This statement, if anything, may be defined as being a gross oversimplification of extraordinarily complex processes. Naturally, pulmonary disease can and frequently does impair breathing, but not necessarily. Very recently a report in this journal illustrated a case where a pathologic condition was noted on a chest radiograph a full decade before the patient became symptomatic.3

I was appalled to read on Page 131 the author’s assertion that, “If you or other team members smoke, that’s acceptable.” The reader who smokes is then admonished not to promote the virtues of non-smoking while smelling of tobacco. I submit that it is completely unacceptable for any health-care worker to smoke!

I bought the book as a potential source for some insights, perhaps experiential, into home care as an adjunct to an outpatient rehabilitation program. I found, instead, 10½ pages of recommendations on collecting data about local home care companies without a clue about using this information in a meaningful fashion. It is of no help whatsoever for those hospitals or agencies entertaining the idea of developing a durable medical equipment (DME) entity of their own.

Pulmonary Rehabilitation/Homecare: From Paper to Practice is a seriously flawed book. I doubt that it would be of any significant value even if it were professionally edited and proofread. In his acknowledgments, Mr Barton castigates the “nattering nabobs of negativism” as being “blithering bozos.” I readily accept the cloak of his colorful alliteration.

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REFERENCES


Knowledge in the basic science aspects of pulmonary medicine has heretofore remained a dispersed entity borrowing from many different disciplines. The book The Lung: Scientific Foundations adopts what appears to this reviewer to be a totally novel approach for textbooks of pulmonary medicine, bringing this knowledge together into one comprehensive compendium.

The stated goal of the book is to “cover the whole field of the scientific foundation of the lung in health and disease including cell biology, biochemistry, morphology, physiology, pharmacology, and general pathological processes.” To achieve this goal, the book assembles an impressive array of 312 contributors, in 30 separate fields, from 17 countries.

One of Descartes’ principal rules in his “Discourse on Method” is, “frequently there is less perfection in a work produced by several persons than in one produced by a single hand.” The cohesiveness of The Lung: Scientific Foundations provides an exception to this rule and is a true testimonial to the superb editorial leadership of Drs Crystal, West, and associates. The unity of the work is maintained by frequent cross-referencing of chapters thereby avoiding duplication of material—a frequent flaw of books of similar format.

The two-volume set is divided into eight sections: an introduction; general cell biologic processes in the lung; major components of the lung; integrated morphology; integrated physiology and pathophysiology; the fetal, perinatal, postnatal, and aging lung; lung injury, defense and repair; and special environments and interventions. Section 5 on integrated physiology and pathophysiology, and Section 8 on intervention are of special interest to respiratory therapists. These two sections contain topics such as: Lung Sounds, Forced Expiration, Gas Exchange, Control of Breathing, Oxygen Therapy and Toxicity, Mechanical Ventilation, and The Lung in the Intensive Care Unit.

The chapters for the most part are easy to read. As expected, some chapters, especially those dealing with theoretical concepts (eg, Kinetics of Oxygen and Carbon Dioxide Reactions and Stress Distribution), appear far too technical and some of the targeted readers may find themselves lacking the background knowledge necessary for an understanding. However, these technical chapters do attempt to strike a balance between the complexity of the subject and mathematical simplicity.

Opposing views and different approaches to a topic are unavoidable.
in medicine. Where applicable, these different perspectives are addressed equally (eg. Design of Airways and Blood Vessels as Branching Trees vs Confluent Trees and Acid Base Physiology). This approach preserves the comprehensive coverage of the work.

Tables and illustrations abound throughout the text, and the chapters are extensively referenced. This contributes to making the book a rich and up-to-date source of information for researchers and clinicians.

This book provides a continuum of knowledge. Although probably not intended for the personal library of the respiratory therapist, The Lung: Scientific Foundations should be included as a reference book in all respiratory care libraries. The book's approach is innovative and should appeal to basic researchers, respiratory care investigators, and clinicians.

Loutfi Sami Aboussouan MD
Fellow
Pulmonary Disease Department
Cleveland Clinic Foundation
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The Flow-Volume Loop—Applied.
Thomas R Gable BA RCPT. Distributed by MedSoft of Vashon, PO Box 13400, Burton Station, Vashon Island WA 98067. Price: $199.99, including shipping. Available for IBM PC or equivalent and Apple II computers.

One of a series of computer-aided instruction programs, this software fills a significant niche in respiratory care education. The program is divided into sections addressing equipment, the flow-volume loop, the breathing maneuver, patient effort, measurements, determination of flow, diagnostic patterns, and review. Each section is accessible from a main menu or the series may be followed through successive sections if the student desires. During a single sitting, the highlighted main menu identifies those sections that have been covered by the student. However, this information is lost once the program is stopped and restarted.

The graphics are simple but functional. Concepts are reduced to their lowest common denominator and then presented with schematic illustrations. I ran this program on a low-quality-graphics portable computer and found it quite adequate. When used with a color monitor, several options for colors are available. Unlike many programs, this software employs oversized type. This makes reading and following the text easy and avoids computer-induced blindness.

Initial setup and loading are simple—even for computer novices. All commands are simple keyboard strokes, and even in playing I was unable to disrupt the function of the program. I did find one technical difficulty. In attempting to run this program from the B drive, I was unable to make it load properly. Even using the DOS trick of assigning the drive to A, I could not overcome the programming difficulty. This may be a problem for those users who have double drives of different sizes and wish to use this program in any but the first or A drive. However, this problem can be overcome by loading the program into the hard drive.

The text comprising this program is concise and easy to understand. Each paragraph contains important information. Almost every section needs to be read, understood, and retained by the student. However, the sections are short enough to be easily and rapidly reviewed at a later time.

After completing the review session, the student is provided with a list of sections to study further if scoring on the multiple-choice questions indicates specific areas of deficiency. This is a strong point of this program.

The lack of built-in questions for the student in each of the sections is a weakness of this program. I believe it would be helpful if students were quizzed as they complete each of the subsections. Useful enhancements would be (1) a mechanism to allow the instructor to independently assess student progress and student comprehension of the material and (2) a bibliography. No references are supplied for the statements made.

This program adequately fulfills its stated purpose, which is to introduce the student to the flow-volume loop in a logical, systematic, and convenient way. Often a difficult subject to teach, this self-paced instructional package can be tailored to each student's learning needs. Its relatively low cost makes it a useful addition to an introductory respiratory therapy library or training program. Although not flashy, its graphics accurately and adequately depict necessary concepts. Because the program is composed of short self-contained segments, students can complete components at their own rate without becoming bored with the overall process.

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Some Possible Misconceptions about Peak Airway Pressures

I was happy to see the editorial by Kacmarek and Hess1 stimulate so much reader response. However, in all the pulmonary-physiology and mechanics crossfire, I note some possible misconceptions. The first is that pressure-controlled inverse-ratio ventilation, or PCIRV, may reduce barotrauma because it reduces peak inspiratory pressure (PIP). One ventilator manufacturer has gone so far as to show a dramatic exploding alveolus in advertisements promoting pressure-controlled ventilation—implying that volume-controlled ventilation is somehow more dangerous because airway pressures increase when lung mechanics change. I believe that this type of advertisement is a disservice to the respiratory care community. The type of misinformation it conveys is reflected in the unsupported statements by some authors (including those responding to Kacmarek and Hess in this journal1†) that lower peak airway pressure (PAP) is good, and, therefore, whatever form of ventilation achieves lower PAP is good. No convincing scientific evidence supports statements like this. And, there are theoretical problems as well.

In the first place, barotrauma is not caused by pressure, despite the literal translation of the term. The disorders associated with barotrauma—pneumothorax, pneumomediastinum, and pulmonary interstitial emphysema—are the result of volume-tric expansion of lung units beyond their elastic limits. This fact has been demonstrated in dogs whose lungs can withstand very large pressures if their chest walls are bound to prevent overexpansion.2,3 We can also invoke the illustration of a toy balloon. If a balloon is normally expanded with 60 cm H₂O pressure, surely it will burst if you try to inflate it to 1,000 cm H₂O. Does pressure cause the balloon to break? No. The balloon bursts because its wall is stretched beyond its elastic limit. If you place the same balloon inside a metal container, it can withstand many thousands of centimeters of water pressure without bursting because the wall of the balloon is prevented from expanding beyond its elastic limit by the metal container. You can argue that it is really transmural pressure that causes the balloon to burst, but this is still mediated by the wall’s elastance. The point is that you can’t talk about barotrauma in terms of pressure alone. You either must talk about pressure and compliance (Δvolume = Δpressure × compliance) or simply about volumetric expansion.

Furthermore, a belief in the advantage of lower PAP seems frequently to be founded on a poor understanding of pulmonary mechanics. It is true that pressure is directly proportional to volume within the lung and hence a good indicator of overdistention and barotrauma. But, PIP during mechanical ventilation is measured at the airway opening, not in the lung. This is a critical distinction because airway pressure at any moment during a mechanical inspiration has two components:

\[
\text{airway resistance} = \frac{\text{volume}}{\text{compliance}} + (\text{resistance} \times \text{flow}).
\]

Thus, PIP is due not only to the delivered tidal volume (\(V_t\)) but also to the inspiratory flow rate at end inspiration for a given compliance and resistance. Peak inspiratory lung pressure is proportional only to \(V_t\).

During volume-controlled ventilation with a rectangular wave form, the flowrate at end-inspiration is the inspiratory flowrate set on the ventilator. PIP is equal to this flow multiplied by the total respiratory system resistance (ie, flow × resistance = pressure) plus the pressure equal to \(V_t\) divided by total respiratory system compliance (ie, volume/compliance = pressure).

In contrast, during pressure-controlled ventilation with a rectangular pressure waveform, flow at end-inspiration is zero. PIP has only the component \(V_t\) divided by compliance (ie, zero flow × resistance = zero pressure). These two situations are illustrated in Figure 1.

It should be clear from this simple analysis that even if pressure-controlled ventilation is set to deliver the same \(V_t\) as volume-controlled ventilation, we expect PIP to be less as a natural consequence of ventilating with a rectangular pressure waveform rather than a rectangular flow waveform. However, the peak alveolar pressure (ie, \(V_t/\text{compliance}\)) during pressure-controlled ventilation is measured at the airway opening, not in the lung. This is a critical distinction because airway pressure at any moment during a mechanical inspiration has two components:

\[
\text{airway resistance} = \frac{\text{volume}}{\text{compliance}} + (\text{resistance} \times \text{flow}).
\]

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It should be clear from this simple analysis that even if pressure-controlled ventilation is set to deliver the same \(V_t\) as volume-controlled ventilation, we expect PIP to be less as a natural consequence of ventilating with a rectangular pressure waveform rather than a rectangular flow waveform. However, the peak alveolar pressure (ie, \(V_t/\text{compliance}\)) during pressure-controlled ventilation is identical to that during volume-controlled ventilation, and the risk of barotrauma is presumably the same. Thus, the fact that PIP is less during pressure-controlled ventilation than during volume-controlled ventilation is totally irrelevant from the standpoint of the patient’s risk of ventilator-induced lung damage. In fact, it could happen that a patient is switched from volume-controlled ventilation to pressure-controlled ventilation at a lower PIP but with a higher tidal volume and thus a higher risk of barotrauma! It depends on how much of a pressure drop is created by the airway resistance.
Of course, these assumptions are based on a simple model of the lungs, and some caveats must be mentioned. If the respiratory system has areas with significantly different time constants, pressure-controlled ventilation may result in better distribution of the $V_T$ among alveolar units. This may place the more compliant areas at lower risk of overdistention.

If ventilation with a rectangular pressure waveform allows more time for alveolar filling, then less of the $V_T$ will be compressed in the terminal airways. This leads, in effect, to a decrease in the dead-space ventilation and an increase in alveolar ventilation. If this occurs, then the delivered $V_T$ can be reduced (ie, by decreasing the PIP) without impairing gas exchange, and, again, the lung is exposed to a lower risk of barotrauma. However, neither of these theoretical situations support the notion that simply lowering PIP, without consideration of all the other factors, is good.

Another misconception is that implied by the statement "When auto-PEEP is generated during IRV, it too should raise (mean airway pressure)." Again, like PIP, mean airway pressure ($P_{aw}$) is measured at the airway opening. It can be shown both mathematically and with a lung model, that $P_{aw}$ is independent of auto-PEEP created by alveolar gas trapping (as distinct from auto-PEEP generated by gas trapped in the ventilator circuit).

A simple ‘thought’ experiment illustrates this. Suppose that a patient is being ventilated with a rectangular pressure waveform with a PIP of 50, PEEP of 10, and inspiratory-to-expiratory-time ratio (I:E) of 1:1 and a frequency of 15 breaths/min. With these settings the $P_{aw}$ is:

$$P_{aw} = (\text{PIP} - \text{PEEP}) \left( \frac{1}{1 + E} \right) + \text{PEEP},$$

or

$$P_{aw} = (50 - 100) \left( \frac{1}{2} \right) + 10 = 30.$$

We assume that the patient’s respiratory system has a relatively short time constant. Because of the low frequency, auto-PEEP (ie, end-expiratory pressure, or EEP) is zero. Now, if the frequency is increased to 60 breaths/min, $P_{aw}$ is still 30 (assuming an ideal ventilator that does not degrade the waveform) but, no doubt, a clinically important level of auto-PEEP is generated in the lungs. In fact, as the frequency increases to infinity, $P_{aw}$ stays constant but the pressure amplitude in the lungs (ie, PIP minus EEP) decreases to zero and the EEP in the lungs (ie, the auto-PEEP level) increases to a near steady-state value equal to $P_{aw}$. The lungs act like a low-pass filter, a device that has actually been used to measure $P_{aw}$.

In summary, I maintain that the concept that barotrauma is caused by pressure without regard to lung mechanics is erroneous. Hence the idea of decreasing the risk of barotrauma by switching from volume-controlled to pressure-controlled ventilation in
an attempt to lower PIP, without regard to changes in the delivered V1 is invalid. Also, PEPaw is a function of the airway pressure waveform parameters (ie, waveform, peak value, baseline value, and I:E) and does not increase as alveolar auto-PEEP increases so long as the airway pressure waveform does not change.

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REFERENCES

Ventilator Circuit Resistance: A Clarification

I find myself in the unavoidably position of responding to my own letter to the editor.1 I trust this will not be interpreted as a precursor to my talking to myself. The purpose of a letter to the editor is to question or expose upon a previous publication, to warn or advise of a finding, or to disseminate information not suitable for an original investigation but important nonetheless. Key to all letters is clarity. I seem to have failed on that score based on comments I have received.

I had intended the letter to warn practitioners not to overlook simple, inexpensive items that may adversely affect the patient despite use of a state-of-the-art, critical care ventilator. Our results demonstrated that a Y-piece configuration available from Marqueta (which we now find is available from several manufacturers) causes significant turbulence at the airway due to its flow-resistive properties. This was demonstrated by the significant pressure drop across the Y-piece in question and its effects on calibration of the Hamilton variable-orifice flow sensor.

Since publication of the letter I have been disturbed by the many misinterpretations of our data. The most disturbing was from a ventilator manufacturer who reportedly used the letter to demonstrate failure of the Veolar to operate properly.

Our further study has shown that use of this Y-piece confounds the pressure monitoring of most ventilators, causing peak pressure to be overestimated and end-expiratory pressure to be underestimated. In fact, our unpublished laboratory findings show that the flow resistance of the Y-piece is great enough to retard normal expiratory flow and create auto-PEEP under certain circumstances.

Our original letter recommends that this particular Y-piece not be used with Hamilton ventilators. I felt this was in line with the Hamilton Operator's manual in which it is recommended to use only "a low-resistance breathing circuit." But I suppose this is a more specific case.

Let me finish by making a broader statement. Minimizing resistance in the breathing circuit decreases the imposed work of breathing and reduces the propensity for development of auto-PEEP. Because this is true, every effort should be made to use a humidifier and ventilator circuit with minimal flow resistance. I suggest, then, that use of the Y-piece in question be avoided regardless of which ventilator is used.

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REFERENCES

Hypnosis Societies

Since the publication of my letter, two hypnosis organizations have changed their addresses and telephone numbers.

Association To Advance Ethical Hypnosis
2675 Oakwood Drive
Cuyahoga Falls OH 44221
Nell Ondorf MA
Executive Director
(216) 923-8880
REFERENCE

AARC & AFFILIATES

August 19-20 in Beachwood, Ohio. The OSRC Continuing Care and Rehabilitation Committee sponsors “The Cardiopulmonary Rehabilitation and Continuing Care Forum.” Topics include pediatric asthma, improving patient motivation and compliance, quality assurance for rehabilitation programs, designing an exercise prescription, reimbursement, and sleep disorders. Contact Lori Kondas at (216) 368-5623.

August 20-23 in Atlantic Beach, North Carolina. The NCSRC hosts its 13th Annual “Symposium by the Sea” at the Sheraton Resort and Convention Center. The keynote lecturer is Dr Dale Oller, a former surgeon to the president of the United States (1985-87). Contact Oral Wise, Wake Medical Center, 3000 New Bern Ave, Raleigh NC 27610, (919) 250-8977.

August 23 in Dayton, Ohio. The OSRC presents the fourth annual Neonatal/Pediatric Conference at Sinclair Center. Contact Susan Mareklinger RRT, St Ann’s Hospital of Columbus, (614) 898-4069.

September 6-8 in Key West, Florida. The FSRC presents its 5th Annual “Southernmost Sunset Seminar” at the Holiday Inn Beachside. Twelve contact hours (AARC/IHS/FBN) are presented, and a sunset dinner/casino cruise held Saturday evening. Contact Dave Robbins, Coral Gables Hospital, 3100 Douglas Rd, Coral Gables FL 33134. (305) 441-6819.

September 11-13 in Lake George, New York. The NYSSRC presents its 12th Annual Symposium, “Roaring Toward Advancement in Respiratory Care: Deficits, Dilemmas, and Decisions.” at the Roaring Brook Convention Center. Speakers include Lorna McBarnette; Paul Mathews; Drs Dolovich, May, Innes, Gluck, Anderson, and Smith; Pat Bowe; and Russ DeTeau. A welcome barbecue dinner, cruise, and Sputum Bowl are planned. Contact Pat Johnson at (518) 374-1651.

September 11-13 in Ocean City, Maryland. The MD/DC Society for Respiratory Care celebrates the 10th anniversary of its Conference at the Carousel Hotel. Meeting topics include neonatal, critical care, pulmonary function testing, home care, rehab, and ethics, along with student and management workshops. Contact Elgloria Harrison at (202) 745-5105.

September 17 in St Louis, Missouri. The Missouri Society District 1 and the Illinois Society Chapter 7 present the Bi-State Pulmonary Conference. Topics include lung transplantation, IVOX, biofeedback, and pediatric respiratory care. A panel discussion is planned on state licensure/registration for respiratory care, with representatives from the Missouri and Illinois Societies for Respiratory Care. For information (daytime), contact John Henkens RRT, Director of Respiratory Care, St Louis University Hospital, (314) 577-8810; or Bruce Mitchell RRT, Director of Respiratory Care, Belleville Memorial Hospital, (618) 233-7750, ext 5564. Evenings, contact Martie Grove CPIT at (314) 869-4125.

September 18-20 in Myrtle Beach, South Carolina. The SCSRC hosts its Annual State Meeting at the Landmark Hotel. Contact Sandra Cassell at (803) 765-7201.

September 19-20 in Sebasco, Maine. The MSRC presents its Annual Fall Conference, “The Maine Event,” at Sebasco Lodge. Topics include case studies of difficult ventilation, hyperbaric medicine, ventilation equipment in the home, and CLIA regulations. Contact Jane Barthelette for additional information at (207) 595-8488.

September 19-20 in Indianapolis, Indiana. The ISRC presents its 18th Annual Fall Seminar at the Holiday Inn-North. The two full days feature lectures, specialty sections, exhibits, and Sputum Bowl competitions. Topics include the future of ventilation, home care accreditation, pediatric rehabilitation, MDI therapy, total quality management, asthma, critical care, and Exosurf use. Contact Kathleen Lee, Ivy Tech, PO Box 1763, Indianapolis IN 46206. (317) 921-4402.

September 25-27 in Minneapolis, Minnesota. The MSRC presents its 22nd Annual Education Conference at the Holiday Inn West. Activities begin with the 3rd annual “Duffers Open” golf tournament and the MSRC “Quiz Bowl.” Dr Ronald Cranford, nationally known biomedical ethicist, gives the keynote lecture. Contact Michele Patnaude at (612) 347-2546.

September 26-27 in Napa, California. The CSRC Chapter 10, along with the American Lung Association of the Redwood Empire and the Respiratory Therapy Program at Napa Valley College, presents the 9th Annual Napa Valley Conference “Current Concepts in Cardiopulmonary Care.” Topics include home care, surfactant replacement therapy, transesophageal echocardiography, AIDS treatments, hyperbaric medicine, and issues in bronchodilator therapy. Also, Patrick Dunne MEd RRT, AARC president, presents “Respiratory Care in the 21st Century.” Nine CEUs. Contact Kate Benscoter at (707) 253-3141.

September 26-27 in Hays, Kansas. The KSRC invites all RCPs to the 5th Annual Western Kansas Seminar. Topics include “RC in Canada,” “Caregiver’s Role in Maintaining Organ Donor Candidates,” “Respiratory Care of the Burn Victim,” “The Lung Cancer Patient,” and “Stabilization/
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Transportation of the Acute Trauma Victim.” Contact Don Hedden RRT, Hays Medical Center-Hadley Campus, 201 E 7th, Hays KS 67601. (913) 628-9310.

October 2-4 in Gaylord, Michigan. The MSRC presents its Annual Fall Conference at the Sylvan Treetops Resort. Program topics include trauma, critical care, aerosol therapy/pharmacology update, pediatrics, management, and ethics. The Pulmonary Rehabilitation Membership Section offers presentations on the metabolic cart and nutrition. Special events include a golf outing on an 18-hole championship course, wine and cheese reception, outdoor steak fry, and volleyball. Contact Beth Hill RRT, Bay Medical Center, Respiratory Care Department, 1900 Columbus Ave, Bay City MI 48708. (517) 894-3166.

October 8-9 in Honolulu, Hawaii. The HSRC presents the 18th Annual Respiratory Conference at the Hilton Hawaiian Village Hotel. Keynote speaker is AARC President Patrick Dunne MEId RRT. Contact Helen Ono, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532.

October 12 in Long Beach, California. The CSRC Chapter IV and the American Lung Association of Long Beach present Respiratory Care Workshop XVII at the Clarion Hotel. Contact Nan Evans, American Lung Association of Long Beach, 1002 Pacific Ave, Long Beach CA 90813-3098. (213) 436-9873.

October 15-17 in Atlantic City, New Jersey. The NJRSC presents the 1991 Shore Conference at Trump Castle by the Bay. Contact Gail Horn RRT, Kennedy Memorial Hospital-Cherry Hill, PO Box 5009, Cherry Hill NJ 08034. (609) 488-6847.

October 18 in Long Island, New York. The Southeastern Chapter of the NYSSRC presents its 23rd Annual Symposium, “Professional Practice Update 1991.” The conference will be held at the Marriott Hotel in Uniondale. Speakers include Patrick Dunne MEId RRT (Continuous Quality Improvement); Marty Douglass MA (Clinical Applications of Noninvasive Ventilation); Barbara Fingure JD (Clinical Incident Documentation); Thomas East PhD (The Use of Computers in Respiratory Care); Aimee Telsy MD (Artificial Surtactant Replacement); and Fred Mindernam BS RRT (Dynamic Interaction Ventilation). Contact Ken Axton RRT at (516) 444-3180.

October 30-31 in Sturbridge, Massachusetts. The MSRC presents its 14th Annual Meeting at the Sheraton Sturbridge Resort and Conference Center. Topics include bronchoscopy, home care, asthma diagnosis and therapy, ECMO, and sleep studies. Special feature is a debate covering the pros and cons of pentamidine and ribavirin. Contact Gina Farquharson RRT, 16 Bartletti St, Pembroke MA 02359. (617) 293-6090.

OTHER MEETINGS

August 29-30 in Kansas City, Kansas. The Respiratory Therapy Department at the University of Kansas Medical Center presents its 23rd Annual Respiratory Care Postgraduate Symposium, “Respiratory Interventions in Trauma Management.” The fee for this two-day program is $50 for AARC members ($10 higher after Aug 23), and the course has been approved by the AARC for 12 Category I credit hours. Contact Homer Rodriguez RRT, Respiratory Therapy Services, University of Kansas Medical Center, 39th and Rainbow Blvd, Kansas City KS 66103. (913) 588-3335.

September 20 in Beaumont, Texas. The Respiratory Care Services Department of St Elizabeth Hospital hosts its 3rd Annual Educational Seminar at the Beaumont Plaza Holiday Inn. Lectures and hands-on workshops cover a wide variety of topics of interest to respiratory and nursing professionals. Contact Greg Rodgers RRT at (409) 899-7065.

September 27 in Philadelphia, Pennsylvania. Main Line Health Inc presents “Current Perspectives in Adult and Neonatal Pulmonary Care” at the Society Hill Sheraton Hotel. Fee: $100 physicians; $75 RTs, RNs, and PAs (includes breakfast and lunch). Hotel and tour packages available. Contact Bob Kay or Bill Howell, Bryn Mawr Hospital, 130 S Bryn Mawr Ave, Bryn Mawr PA 19010. (215) 526-3340.

September 27 in Oxnard, California. The American Lung Association of Ventura County presents “Pandora’s Box of Pulmonary Medicine.” This year’s guest speaker, Thomas Petty MD, discusses pulmonary rehabilitation. Contact Barbara Weinberg, American Lung Association, (805) 988-6023.

October 11-13 in Clearwater Beach, Florida. The American Lung Association of Florida hosts the conference “Respiratory Care for the Pediatric and Neonatal Patient.” Contact Richard T Doggett, American Lung Association of Florida, PO Box 8127, Jacksonville FL 32239. (904) 743-2933.

October 11-13 in Marina del Rey, California. Children’s Hospital of Los Angeles, Saddleback Memorial Medical Center, and SensorMedics Corp host the Infant and Pediatric Pulmonary Function Testing Conference. For a brochure and more information, call (800) 234-2466, ext 700.

January 20-27 in Bahamas Cruise/orlando Tour. Dream Cruises’ “Stress Busters Cruise” for continuing education will combine lectures and workshops throughout the week. $795 (per person/double occupancy) inside cabin or $825 (per person/double occupancy) outside cabin. Price includes airfare, cabin, hotel, car, transfers, with food and entertainment provided aboard ship. Contact Kathy Kearney at (800) 462-3628, or write: 10882 LaDona Ave, Garden Grove CA 92640.
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Item CN7 — $8 Ea

Toxicity of Solutions and the Respiratory Tract
This package will give you an understanding of how to treat those who have received toxic respiratory care. Includes instructions on the treatment of various respiratory tract pathologies.
Item CN8 — $6 Ea

Carbon Monoxide Inhalation: Introduction to Physiologic Effects and Respiratory Management
This package explains how carbon monoxide inhalation affects oxygen transport mechanisms in the body and the use of CO as a diagnostic test for pulmonary function. Also covers recognition signs and symptoms, and treatment of CO poisoning, including the equipment needed to administer therapy.
Item CN9 — $7 Ea

Electrical Safety in Respiratory Therapy I: Basic Electrical Circuitry
Enables you to relate the basic principles of electrical theory to commonly used equipment and procedures. Includes an understanding of electrical safety in respiratory care.
Item CS12 — $6 Ea

Electrical Safety in Respiratory Therapy II: Identification of Electrical Hazards
After using this IISP, you will be able to identify and, where possible, minimize or eliminate electrical hazards in the hospital. It will help you differentiate between the different types of electrical hazards to which patients and hospital personnel are exposed. Then you learn how to implement the basic principles of electrical safety in the hospital.
Item CS13 — $6 Ea

Microbiology for Respiratory Therapy: A Review of Microbial Growth and Contamination
Provides you with an overview of some important aspects of microbiology in respiratory care. Includes many topics such as groups and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of transmission.
Item CS17 — $9 Ea

Patient Evaluation

Pulmonary Function Assessment I: Basic Screening Studies
Helps you understand the results of simple spirometry tests and gives the definition of obstructive and restrictive pulmonary disorders and descriptions of the basic pathology involved in each. It further covers the four reasons for simple spirometry tests, the common parameters of lung function measured by simple spirometry, and helps you identify the results from spirometry indicative of obstructive and restrictive pulmonary disorders.
Item PE3 — $8 Ea

Pulmonary Function Assessment II: Bedside Studies
This study unit is designed to familiarize you with the use and interpretation of bedside pulmonary function tests. You will learn how pulmonary function test results can be used to assess respiratory function and which tests are commonly used. Also, learn what measuring instrument is required for each test, how the test is performed, and what is significant about the test results.
Item PE4 — $7 Ea

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies
This material is designed to help you recall the eight subdivisions of total lung volume and their individual characteristics in health and disease. Also, helps you understand the measurement of lung volume by nitrogen washout, helium dilution, and closing volume techniques.
Item PE5 — $6 Ea
Body Plethysmography
Explains the theory of body plethysmography and its use in the measurement of FRC and airway resistance. Defines Boyle's Law, thoracic volume, airway reaction, and its normal values. Also describes the measurement of airway resistance using body plethysmography.

Item PE6 — $9 Ea

Sources of Error in the Determination of Blood Gas Values and pH
This study package will familiarize you with the errors that may occur in the analysis of blood gases and pH. These are often the most important laboratory data used in the diagnosis and treatment of pulmonary disease, and errors in these values can result in deleterious effects on patient care.

Item PE7 — $10 Ea

Temperature Adjustment of Blood Gases and pH
The principal goal of this study package is to teach you the effects of abnormal body temperature on blood gas and pH values. Blood gas values and pH are determined at 37 degrees Celsius, but if a patient's temperature is abnormal, the in vivo blood gas and pH values will be different from those reported by the blood gas laboratory. This package will teach you how to adjust these values.

Item PE8 — $10 Ea

Measurement of Static Compliances and Dynamic Characteristics Curves During Mechanical Ventilation
Upon completion of this package, you will be able to compute and record static compliance curves and dynamic characteristics. You will also be able to describe the procedure of obtaining pressure-volume measurements and be able to interpret the compliance characteristics measurements.

Item PE9 — $7 Ea

Articular Blood Gas Interpretation
Teaches you an understanding of ABG interpretation in order that the therapy prescribed by the physician can be administered in a knowledgeable manner. A systematic method will be described that allows you to correctly classify the acid-base dysfunction and to relate the diagnosis concisely and coherently to other members of the health care team.

Item PE10 — $10 Ea

Clinical Practice
Chest Tubes and Pleural Drainage
Helps you understand the purpose of pleural drainage, how it might affect the patient's respiratory status, and what precautions you must take when working with patients who are receiving pleural drainage.

Item CP3 — $6 Ea

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation
After completing this ISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.

Item CP4 — $6 Ea

Tracheal Intubation II: Routes of Intubation
Describes the four routes of tracheal intubations and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.

Item CP5 — $6 Ea

Tracheal Intubation III: Equipment Procedures for Intubation
 Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.

Item CP6 — $8 Ea

Respiratory Management of Neuromuscular Crisis
This ISP will be helpful in the respiratory management of patients with ventilatory failure caused by a neuromuscular disorder. It will provide you with a basic understanding of how neuromuscular conditions lead to ventilatory insufficiency and what special considerations must be taken when working with these types of patients.

Item CP7 — $7 Ea

Respiratory Management of Flail Chest
This study package helps you increase your understanding of the pathophysiology of flail chest and the respiratory management of patients who have sustained chest wall trauma that results in a flail chest.

Item CP8 — $7 Ea

Respiratory Management of Head Trauma
This ISP teaches you to identify five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, PO2, and PCO2.

Item CP9 — $7 Ea

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1. $2,000 for the best original paper (study, evaluation, or case report) accepted for publication in RESPIRATORY CARE from November 1990 through October 1991.

2. Four awards of $1,000 each for papers based on 1990 OPEN FORUM presentations accepted for publication in RESPIRATORY CARE from November 1990 through October 1991.

3. Five awards of $500 each for the best papers (submitted, not necessarily published) from 'never published in RC' OPEN FORUM participants. The author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the Annual Meeting (received in the Editorial Office by December 1).

REGISTRATION REIMBURSEMENT FOR OPEN FORUM PAPERS

Any 1991 OPEN FORUM presenter (or co-author designate) who submits an adequately prepared paper based on his or her Open Forum presentation prior to or at the 1991 Annual Meeting will be reimbursed for Annual Meeting registration. The submitted paper must contain complete data, be appropriately organized, and be typed double-spaced. Camera-ready artwork need not be submitted with the paper, but sketches of proposed figures should be clear and detailed enough to allow critique.

AARC ANNUAL CONVENTION SITES & DATES

1991—Atlanta, Georgia, December 7-10
1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994—Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5

RADIOMETER AMERICA

Radiometer America Inc is offering three awards of $333 each for the best features from (1) Test Your Radiologic Skill, (2) Blood Gas Corner, and (3) PPT Corner accepted for publication from November 1990 through October 1991. The awards will be made at the 1991 Annual Meeting. Papers are judged automatically. No application is necessary.

THE NATIONAL BOARD FOR RESPIRATORY CARE

1991 Examination and Fee Schedule

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<td>Entry Level CRRT—new applicant: $75.00</td>
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<td>Written Registry Only reapplicant: $50.00</td>
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<td></td>
<td>Clinical Simulation Only new and reapplicant: $100.00</td>
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<td></td>
<td>Entry Level CPFT—new applicant: $100.00</td>
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<td></td>
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1. **PRODUCT IDENTIFICATION:**

- Name of Product and Type of Device
  (Include sizes or other identifying characteristics and attach labeling, if available)

- Manufacturer's Name

- Manufacturer's City, State, Zip Code

- Lot Number(s) and Expiration Date(s) (If applicable)

- Serial Number(s)

- Manufacturer's Product Number and/or Model Number

- Is this a disposable item? YES ☐ NO ☐

2. **REPORTER INFORMATION:**

- Your Name __________________________ Today's Date __________________________

- Title and Department __________________________

- Facility's Name __________________________

- Street Address __________________________

- City __________ State __________ Zip __________ Phone ( ) ___________ Ext: __________

3. **PROBLEM INFORMATION:**

- Date event occurred __________________________

- This event has been reported to: Manufacturer ☐ FDA ☐

- Please indicate how you want your identity publicly disclosed:
  - No public disclosure ☐
  - To the manufacturer/distributor ☐
  - To the manufacturer/distributor and to anyone who requests a copy of the report from the FDA ☐

- If requested, will the actual product involved in the event be available for evaluation by the manufacturer or FDA? YES ☐ NO ☐

- Problem noted or suspected (Describe the event in as much detail as necessary. Attach additional pages if required. Include how and where the product was used. Include other equipment or products that were involved. Sketches may be helpful in describing problem areas.)

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Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that “We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication.”

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is unique or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author’s personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving inappropriately or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author’s & Typist’s Kit

To discuss a writing project, write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author’s & Typist’s Kit. The Kit provides authors with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. Typists can use the Kit’s Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—Typist

• Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
• Number pages in upper right corner and leave margins of 1½” or more on all four sides of the page.
• For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
• Meticulously follow instructions for typing references.

General Concerns—Author:

• Structure manuscript as specified hereafter.
• Provide all requested information on title page as specified hereafter.
• Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
• Consider having paper reviewed in-house before submission.
• Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer’s name & affiliation, references. Tables & figures may be included. Double-space everything. Write “For Publication” on title page.

Structure: Important Details

Title Page: List title of paper, all authors’ full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name source of grants or other support. Identify any author’s consulting or commercial relationships that pertain to the paper’s topic.
INSTRUCTIONS FOR AUTHORS & TYPISTS

Abstract Page: Number this Page 1. List paper’s title but omit authors’ names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 x 7 to 8 x 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but rough sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author’s name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder’s written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, “PEEP, 10 cm H2O [0.981 kPa].” For conversion to SI, see Respiratory Care 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer’s name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parenthases. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:
• Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
• Cite only published works— or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal’s name, followed by “(in press).”
• In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
• In the reference list, place the cited works in numerical order.
• For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the original cited articles and books, not from secondary sources such as other articles’ reference lists, which often are inaccurate.
• Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in Index Medicus.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

Corporate Author Journal Article:

Article in Journal Supplement:
(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

Abstract in Journal:
(abstracts are not strong references: when possible, full papers should be cited. When cited, abstracts should be identified as such.)

Editorial in Journal:

Letter in Journal:

Personal Author Book:


Corporate Author Book:

Book with Editor, Compiler, or Chairman as ‘Author’:

Chapter in Book:

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to Respiratory Care, 11030 Ables Lane. Dallas TX 75229 (or Federal Express to Respiratory Care, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under General Requirements at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author’s Checklist
1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
3. Is title page complete?
4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
7. Have SI values been provided?
8. Has all arithmetic been checked?
9. Has manuscript been proofread by all authors?
News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to Respiratory Care Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229.

AIRWAY PRESSURE MONITORS. The Ventilarm II (Cat No 5622) and Remote Alarm (Cat No 5624) are designed to continuously monitor airway pressure in adult and pediatric ventilator circuits. According to the manufacturer, the Ventilarm II is well suited for static pressure applications (PEEP) and communicates over standard phone cable to the Remote Alarm up to 200 feet away. Both units can be powered by AC or DC current. Hudson RCI, Dept RC, 27711 Diaz Rd, PO Box 9020, Temecula CA 92390-0740. (800) 848-3677.

BLOOD SAMPLING SYSTEM. The new Safedraw Closed-loop Blood Sampling System is designed to minimize the risks associated with arterial-line blood sampling. According to the manufacturer, the shielded, blunt plastic Safe Needle makes needle sticks virtually impossible; the self-sealing blood-sampling septum ensures no non-sterile contact with the fluid path and prevents blood spillage; and the permanently attached volume-restricted syringe aspirates and holds the saline-diluted blood within pressure monitoring tubing, consistently ensuring pure samples and allowing for refusion of otherwise discarded blood. The preconnected system is designed to add very little componentry by utilizing existing pressure tubing to hold saline-diluted blood. Viggo-Spectramed, Dept RC, 1900 Williams Dr, Oxnard CA 93030-2691. (805) 983-1300.

PULSE OXIMETER. The MiniOX V pulse oximeter is a portable, 14-ounce instrument capable of monitoring SaO2 and pulse rate on difficult-to-monitor patients such as neonates and adults with low perfusion, according to the manufacturer. One standard 9-V battery provides power for up to 200 hours of continuous operation. The MiniOX V features an easy-to-read LCD, an audible signal with each heartbeat, and user-programmable audible and visual alarms for high and low readings. Finger-clip, flex, and neonate sensors are available; and a rear bracket and universal mounting clamp allow the MiniOX V to be attached to bed rails, ventilator arms, and I.V. poles. The MiniOX V also can be connected to the new pocket-size MiniOX printer. MSA Catalyst Research, Dept RC, 3706 Cordonall Ln, Owings Mills MD 21117. (800) 851-4500 or (301) 356-2400.

BLOOD GAS ANALYZER. The ABL510 is the first blood gas analyzer capable of determining both acid-base and oxygen status from a single analysis, according to the manufacturer. The ABL510 is capable of measuring total hemoglobin, oxygen saturation, pH, Po2, and PCO2, and is capable of calculating up to 40 derived variables covering patient oxygen and acid-base status, pulmonary function, hemoglobin-oxygen affinity, metabolism, and arterial oxygen transport capacity. The ABL510 requires only an 85 μL blood sample and provides a report in as little as 45 seconds. Radiometer America Inc, Dept RC, 811 Sharon Dr, Westlake OH 44145. (800) 736-0600 or (216) 871-8900.
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Proventil
(albuterol sulfate, USP)
Solution for Inhalation

Unit Dose 0.083%* 0.5% 20 mL bottle
*Potency expressed as albuterol.

DESCRIPTION: Albuterol sulfate is a beta-2-adrenergic bronchodilator (see Clinical Pharmacology). Inhalation solution contains albuterol sulfate in a white crystalline powder, soluble in water and slightly soluble in ethanol. The intravenous generic name for albuterol base is salbutamol.

PROVENTIL Solution for Inhalation is available in two concentrations. The 0.5% solution is in concentrated form (0.5 mL of the solution to 3 mL of normal saline solution prior to administration). The 0.083% solution reconstitutes in 10 mL of normal saline solution.

Each mL of PROVENTIL Solution for Inhalation (0.5%) contains 5 mg of albuterol base (1.84 mg albuterol sulfate) in an isotonic aqueous solution containing 0.08% acetic acid, 10 mg of sodium citrate dihydrate, 95 mg of sodium chloride, 72 mg of kaolin, 0.001 mg of sodium bromide, and 0.0005 mg of sodium benzoate. Each mL of PROVENTIL Solution for Inhalation (0.083%) contains 0.5 mg of albuterol base (0.16 mg albuterol sulfate) in an isotonic aqueous solution containing sodium chloride and boric acid buffer to adjust the pH to 5.5. The 0.083% contains no sodium benzoate. It is supplied as 3 mL units for metered-dose dispensers.

PROVENTIL Solution for Inhalation is a clear, colorless to light yellow solution.

CLINICAL PHARMACOLOGY: The prime action of beta-adrenergic drugs is to stimulate adrenergic endplates. The catecholamines which constitute the family of cyclic-3',5'-adenosine monophosphate (cyclic AMP) from adenosine monophosphate (AMP). The cyclic AMP thus formed mediates the cellular responses in vivo and vitro,
THE ONLY UNIT DOSE ALBUTEROL SULFATE

IT'S THE EASY SOLUTION

Proventil (albuterol sulfate, USP)
Solution for Inhalation
Unit Dose 0.083%*
0.5%* 20 mL bottle
*Potency expressed as albuterol

Please see full prescribing information on adjacent page.

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